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Teneille R. Brown, JD

Abstract

The opioid addiction epidemic is the most overwhelming public health crisis our country has faced. It is now creating a legal crisis, as the its poisonous fruits spill over into the criminal, tort, and family courts. The epidemic costs the U.S. economy about $500 billion every year, and the pressure is crippling our legal systems. This Article is an attempt to relieve some of that pressure, by advocating for a comprehensive public health campaign based upon a new model of addiction. Research shows that the prevalent “moral choice” model of addiction has facilitated stigma and discouraged treatment, by viewing affected individuals as blameworthy, different in kind, and hopeless. Even when programs are accessible, which they often are not, individuals will not seek treatment because they fear adopting the label of “addict.” In this Article, I affirmatively reject the moral choice model, identifying it as an obstacle to mitigating the opioid epidemic. In its place, I offer a model of addiction that more closely tracks its complex disease etiology, while humanizing people with addiction, removing stigma, and encouraging treatment. I refer to this model as the “integrated disease model,” or IDM, as it explains addiction as a neuro-genetic phenomenon, but does not locate addiction entirely in the brain. Rather, it places addiction on equal footing with other chronic diseases, such as lung cancer or diabetes, each of which has significant genetic, behavioral, and environmental causes. This Article will explain 1) how the moral choice model leads to no treatment and poor treatment, 2) how the law has furthered stigma through the criminalization of addiction, 3) and why we need to fund a comprehensive public health campaign based upon findings from neuro-genetics and public health. The IDM emphasizes the biological continuum of genetic risk factors to which we are all susceptible, the neurological networks that are impaired once the addiction has taken hold, and finally, the incredible power of evidence-based treatments. Explaining addiction in this way—as a treatable, complex disease—has been shown to reduce stigma and encourage treatment.

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ADDITION AS DISEASE

INTRODUCTION

A. The Problem of Addiction Stigma

Stigma is a powerful and multidimensional social force. It can be based on visible markings like skin color or leprosy, or it can be based on invisible characteristics such as being widowed or mentally ill. Essentially, stigma is defined as labeling differences in others, then using those differences to reduce someone from “whole and usual” to “tainted and discounted.” Stigma must be understood with reference to a power structure, as it reproduces inequities among under-privileged groups. Like stereotyping, the process of stigmatization relies on sticky overgeneralizations, which in turn are used to justify social exclusion, prejudice, and discrimination. Stigma manifests in three distinct ways. It first develops between members of a society through gossip and social sanctions, then may become manifest in legal and social institutions such as housing and employment, and eventually is internalized by those stigmatized, leading to shame and reduced feelings of self-worth.

Drug addiction is extraordinarily stigmatized. A comprehensive study by the World Health Organization found that drug addiction ranked at the top of a list eighteen stigmatized social problems. Among mental illnesses, which as a group are quite stigmatized, drug addiction ranks as the most stigmatized disorder, with lay people rating “addicts” as more dangerous, less predictable, and more to blame for their disorders than

1 Bernice Pescosolido, The Public Stigma of Mental Illness: What do we think; what do we know; what can we prove? 54 J. HEALTH AND SOCIAL BEHAVIOR 1, 3 (2013)
2 Laramie Smith, et al., Substance Use Stigma: reliability and validity of a theory-based scale for substance-using populations 162 DRUG ALCOHOL DEPENDENCE 34, 36
people with depression or schizophrenia.\(^6\) With blame comes moral judgment.\(^7\)

Because the word “addict” carries with it such negative connotations, and conflates the disorder with the individual, the preferred convention now is to refer to addicts as people with substance use disorder, or SUD. Studies have shown that the way we talk about addiction matters, and can exacerbate stigma.\(^8\) However, as this article is exploring the very underpinnings of these negative connotations and the social response to the label of “addict”, I will sometimes employ the non-clinical and stigmatized terminology.

The predominant narrative of addiction holds that it is caused by a weak character and immoral choices. Media portrayals rely on this model, with ubiquitous stories of individual addicts who made bad decisions, broke the law, and never obtained sobriety. This “moral choice” model has facilitated stigma, by treating affected individuals as blameworthy, different in kind, and hopeless. Coincidentally, these same attributes are frequently used to dehumanize individuals, and to justify social distance, harsh punishment and legal discrimination. Stigma associated with the moral choice model has discouraged many people from getting treatment, as they resist adopting the label of the “addict” even when their disorder is advanced. Quite literally, stigma kills.\(^9\)

As addiction manifests in socially undesirable behavior, judgment toward people with SUD is understandably complicated. People with SUD may deceive and manipulate to obtain drugs or hide use. Erecting healthy boundaries with someone with SUD is not enacting stigma. This may be necessary self-protection. We can, however, discourage the unhealthy and antisocial behavior, without shaming the individual by helping these individuals seek clinical treatment, and understanding that their decisions are highly constrained by a powerful disease. Frankly, we must, re-humanize addiction by treating addiction like any other complex, and


\(^7\) M.C. Angermeyer and Sandra Dietrich, *Public Beliefs about and Attitudes Towards People with Mental Illness: a review of population studies*, 113 Acta Psychiatr. Scand. 163, 170 (2006); “[Our research showed] that people addicted to drugs were viewed as significantly more responsible for their disorder compared to people with mental illness or those in a wheelchair…blame [led to perceptions] that they were most able to overcome it.”

\(^8\) For example, when physicians received a vignette that described the patient as a “substance abuser,” as opposed to “someone with substance abuse disorder,” they were more likely to blame the individual for his problem, and think he should be punished for not adhering to court-ordered treatment. See, Harvard Mental Health Letter, Harvard In-brief, April 2010, available online at http://www.health.harvard.edu/newsletter_article/addiction-terminology-affects-clinicians-attitudes-towards-patients.

chronic disease. Once addiction is properly conceived of as a disease, we can then make sure that people with SUD have access to treatment, so they can stop the cycle of antisocial behavior. Just as people with bipolar disorder or depression may at times seem “selfish” or difficult to handle, we should not use their untreated behavior as a justification for denying them treatment.

In this Article, I affirmatively reject the moral choice model. In its place, I offer a model of addiction that more closely tracks its disease etiology, while humanizing people with addiction, removing stigma, and encouraging treatment. I refer to this model as the integrated disease model, or IDM, as it explains addiction as a neuro-genetic phenomenon, but does not locate addiction entirely in the brain. Rather, it places addiction on equal footing with other complex and chronic diseases, such as lung cancer, or diabetes, which also have significant genetic, behavioral, and environmental causes. Employing the IDM, I advocate for a massive, new, federal public health campaign that uses neuro-genetic findings and stories of recovery to demonstrate that people with addiction are not blameworthy, not different in kind, and not hopeless.

By emphasizing that addiction is a disease, and thus prioritizing prevention and treatment of affected individuals first and foremost, we can hopefully reduce the unfair stigma that hinders recovery and leads to antisocial behaviors. After clinical care is prioritized, we can then work on social policies that promote inclusion, such as criminal justice reform and fair housing and employment practices. But a key premise of this Article is that we ought not “jump the gun” to focus on social determinants of health, when we have not even adopted a model of addiction that prioritizes the primary determinants of health.

This Article will thus proceed in four brief parts. In the first part, I will provide evidence for the existence of stigma toward people with SUD, and explain how it leads to no treatment and poor treatment. In the second

10 Lung cancer and diabetes can be caused by personal choices and behavior, such as smoking cigarettes, eating too much sugar, or using intravenous needles. So too can addiction be caused in part by voluntary choices to consume drugs. There are also genetic and biological vulnerabilities to lung cancer and diabetes, just as there are with addiction, that can exacerbate risk and make conditions worse. Given that Type 2 diabetes is sometimes caused by eating too many carbohydrates and sugars, it remains stigmatized, even with its clear adoption as a medical disease. The IDM will not remove all stigma. See generally, Jessica Browne, et al., ‘I call it the blame and shame disease’: a qualitative study about perceptions of social stigma surrounding type 2 diabetes, 18 BMJ Open e003384 (2013)Without delving too far into the sociology of health, there are of course difficulties distinguishing between an illness (where a person experiences symptoms) a disease (where someone calls for professional help) and a sickness (where the person adopts the social role of patient). However, the disease model is used as a heuristic to explain a medically-oriented model, that encourages reflection on the biological and environmental causes of disease. See, Anna-Henriikje Seidlein and Sabine Salloch, Illness and disease: an empirical-ethical viewpoint, 20 BMC MEDICAL ETHICS 5, 5 (2019)
part, I will explain the law’s role in furthering stigma through the criminalization of addiction, and our ineffective efforts to reduce stigma through statutes like the Americans with Disabilities Act. In the third section, I will explain competing models of addiction, providing evidence for a disease model based on neuro-genetic causes and effects. In the fourth section, I conclude with my recommendation for a new public health campaign, based upon the IDM and the neuro-genetic risks of addiction. As compared to previous models of addiction, which have focused attention either exclusively on choice or the brain, this model incorporates aspects of both to treat addiction like other chronic diseases. Employing the IDM, I advocate for a massive, new, federal public health campaign that uses neuro-genetic findings and stories of recovery to demonstrate that people with addiction are not blameworthy, not different in kind, and not hopeless.

B. Why This Matters: The Opioid Crisis is a Public Health Emergency

At the same time as physicians were recognizing rampant undertreatment of pain, pharmaceutical companies in the 1990s began aggressively marketing new opioid medications that they claimed were unlikely to be abused. This proved to be fatally, fraudulently, wrong. Consumption of oxycodone increased by a whopping 500% from 1999 to 2011, and opioid-related overdoses almost quadrupled. The alarming increase in opioid use has led to the “worst drug overdose epidemic in [US] history,” according to the Centers for Disease Control.

Overdose deaths from opioids have led to an absolute decline in life expectancy in the United States, and it is the number one cause of accidental death. In 2016, the overdose death rate from synthetic opioids doubled from the last year, likely driven by an influx of potent, non-

“A consistent feature in [Purdue Pharmaceutical’s] promotion and marketing of OxyContin was a systematic effort to minimize the risk of addiction in the use of opioids for the treatment of chronic non–cancer-related pain.”
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prescription fentanyl from China. More than a hundred people continue to die from the epidemic every day. This has led the Department of Health and Human Services and the President of the United States to declare our modern addiction crisis a “public health emergency.”

The opioid crisis has exposed a devastating reality: with the right combination of environmental stress and genetic vulnerability, any of us could become addicted to drugs. Any of us could start taking OxyContin for kidney stones or a sports injury, and end up living on the streets after our family has kicked us out. Any of us could die of a heroin or fentanyl overdose. A big part of what separates those who become addicted from those who do not is something entirely outside of our control: our genes.

Many who have been touched by the opioid crisis now appreciate that anyone can become addicted, regardless of race or socio-economic status. However, stigma is still felt by people with opioid use disorder (OUD), and disproportionately so for those from lower social classes. Indeed, social class is a better predictor of adverse outcomes from addiction than the patterns or volume of drug use itself. While people who experience childhood trauma are at an increased risk of developing SUD, addiction is still addiction when it manifests. And it is still largely viewed as a disease of the morally bankrupt or the weak-willed. We see this in the

16 See, Centers for Disease Control Newsroom, U.S. drug overdose deaths continue to rise; increase fueled by synthetic opioids, March 29, 2018, available online at https://www.cdc.gov/media/releases/2018/p0329-drug-overdose-deaths.html ("Across demographic categories, the largest increase in opioid overdose death rates was in males between the ages of 25-44."); see also, Colleen Barry, Fentanyl and the Evolving Opioid Epidemic: what strategies should policy makers consider? 69 PSYCHIAT. SERV. 100, 100 (2018) (encouraging a public health messaging campaign that educates on the risks of fentanyl).

17 Health and Human Services, What is the U.S. Opioid Epidemic?, HTTPS://WWW.HHS.GOV/OPIOIDS/ABOUT-THE-EPIDEMIC/INDEX.HTML, UPDATED JAN. 2019

18 “On October 26, 2017, President Trump announced that his Administration was declaring the opioid crisis a national Public Health Emergency under federal law, effective immediately. ‘I am directing all executive agencies to use every appropriate emergency authority to fight the opioid crisis,’ the President said.” See, The White House, The Opioid Crisis, available online https://www.whitehouse.gov/opioids/

19 Shelly Wiechelt & Shulamith Lala A. Straussner, Introduction to the Special Issue: Examining the Relationship Between Trauma and Addiction 15 J. SOCIAL WORK PRACTICE IN THE ADDICTIONS 1, 1 (2015) ("Furthermore, it is empirically well established that there is a link between trauma-related disorders and sub- stance use disorders.") See also, Annett Lotzin, et al., Profiles of Childhood Trauma in Patients with Alcohol Dependence and Their Associations with Addiction-Related Problems, 40 ALCOHOLISM: CLINICAL AND EXPERIMENTAL RESEARCH 543, 543 (2016)“The high occurrence of childhood trauma in individuals with alcohol dependence is well-recognized.”

20 Nora D. Volkow and A. Thomas McLellan, Opioid Abuse in Chronic Pain — Misconceptions and Mitigation Strategies 374 NEW ENGL. J. MED. 1253, 1257 (2016)

21 Robin Room, Stigma, Social Inequality and Alcohol and Drug Use, 24 DRUG AND ALCOHOL REVIEW 143, 143 (2005)

22 Shelly Wiechelt & Shulamith Lala A. Straussner, Introduction to the Special Issue: Examining the Relationship Between Trauma and Addiction 15 J. SOCIAL WORK PRACTICE IN THE ADDICTIONS 1, 1 (2015)
way that people think addiction should be treated, which is often through peer-counseling, jail time, and cold-turkey abstinence, rather than through a health care clinic.  

I. EVIDENCE OF ADDICTION STIGMA

It is perhaps no surprise that people with SUD are extensively stigmatized. However, the extent of the stigma is a bit astounding. Widespread stigma presents at the social, structural, and personal levels. In this section I will analyze the evidence for each. Given how frequently people hold stigmatized views of addicts, and how this leads to massive under-treatment, it is disconcerting that fewer than ten experimental studies exist on SUD stigma. Despite the clear evidence we have of the existence of stigma from observational studies and surveys, more empirical research needs to be done on how best to mitigate it.

a. Social Stigma

We Americans report wanting considerable social distance from people with SUD. People with SUD are rated as having little social value, and therefore it is considered acceptable not to help them, and to exclude them from social spaces. For example, 75% of Americans are unwilling to have someone with drug dependence move next door to them. Nearly 60% of Americans would be unwilling to make friends with someone with "drug dependence," and nearly 73% would be unwilling to even spend one evening socializing with an addict. Let that sink in. Given this extreme desire for social distance, it is no wonder that 90% of respondents say they are unwilling to have someone with drug dependence marry into their family. Seventy-eight percent say they are unwilling to have someone with drug dependence marry into their family. Seventy-eight percent say they are unwilling to have someone with drug dependence marry into their family.


Patrick Corrigan & Katherine Nieweglowski, Stigma and the public health agenda for the opioid crisis in America, 59 INTL. J. OF DRUG POLICY 44, 44 (2018)

Patrick Corrigan, et al., The Public Stigma of Mental Illness and Drug Addiction: Findings from a Stratified Random Sample, 9 J. SOC. WORK 139, 139 (2009); see also Kumiko Yoshioko, et al., Associations between Beliefs about the Causes of Mental Disorders and Stigmatizing Attitudes: Results of a Mental Health Literacy and Stigma Survey of the Japanese Public, 45 INT. J. MENTAL HEALTH 183, 183 (2016); Shuntaro Ando et al, Review of mental-health-related stigma in Japan, 67 PSYCHI CLIN. NEUROSCL. 471, 471 (2013)

Bernice Pescosolido, The Public Stigma of Mental Illness: What do we think; what do we know; what can we prove? 54 J. HEALTH AND SOCIAL BEHAVIOR 1, 9 (2013)

Bernice Pescosolido, The Public Stigma of Mental Illness: What do we think; what do we know; what can we prove? 54 J. HEALTH AND SOCIAL BEHAVIOR 1, 9 (2013)
drug dependence work closely with them on their job.\textsuperscript{28} These levels of stigmatizing attitudes are quite a bit higher than for people with depression or schizophrenia.\textsuperscript{29}

The little research on stigma that is specific to OUD reveals that the stigma persists in the face of our recent crisis. Many thought that stigma might be lessened. Unlike heroin or cocaine, which are illegal, OUD could have begun with a valid prescription from a doctor, even if the pills were later diverted to someone without a prescription. Some thought this might reduce the moral judgment against people with OUD, given its ambiguous legal status.\textsuperscript{30} It was also thought that because the people affected by OUD were more likely to be white, wealthy, and have insurance, compared to those impacted by the previous addiction crises (such as the cocaine epidemic of the 1980s and 90s), people may have less stigmatizing attitudes toward them.

So far, this has not turned out to be the case. The prevalence of OUD is \textit{not} decreasing many forms of stigma. Respondents with personal experience with someone with OUD were \textit{more likely} to say that 1) people with OUD are to blame for their disorder, 2) some people lack the self-discipline to use pain medications responsibly, and 3) and employers and landlords should be allowed to deny employment or housing to people with OUD.\textsuperscript{31} Fewer people are unwilling to have someone with OUD work closely with them (59\%) or marry into the family (66\%), compared to previous studies of drug addiction generally.\textsuperscript{32} However, the desire for social distance remains quite high in the general population, and in some cases is slightly \textit{higher} for people who have personal experience with someone with OUD. Greater exposure to people with SUD is not going to be the silver-bullet to reducing stigma. This might be due to the manipulative behavior that these friends have experienced. They are not discriminating based upon ignorance of addiction; they are discriminating based upon perceived behavior. This must be addressed at the systemic level, by creating effective treatments and improving access to them, and by removing the stigma associated with being someone with SUD.

Most Americans believe that people with SUD are dangerous and


\textsuperscript{29} Bernice Pescosolido, \textit{The Public Stigma of Mental Illness: What do we think; what do we know; what can we prove?} 54 J. HEALTH AND SOCIAL BEHAVIOR 1, 9 (2013)

\textsuperscript{30} Kennedy-Hendricks, et al, 68 Psychiatric Serv.462 (2017)


unpredictable.\textsuperscript{33} Despite evidence that people with SUD are more likely to injure themselves, many Americans believe that persons with alcohol or drug addiction are more likely to be violent toward others.\textsuperscript{34} This is likely due to the depiction of people with drug addiction in the mass media and popular culture.

Our knowledge about persons with addictions is shaped through the “visible, marginalized street populations of persons with addictions, or through stereotypes of persons with addictions as portrayed in movies.”\textsuperscript{35} Unfortunately, people with SUD are represented in film in stereotypical ways that do not reflect their diverse lives.\textsuperscript{36} Society has become inured to viewing portrayals of untreated people with SUD as “disheveled, often homeless, and potentially dangerous.”\textsuperscript{37} We need to depict people with SUD in more mainstream, and diverse, ways.\textsuperscript{38}

Individuals with substance use disorders elicit great social distance across all stakeholders. The public, family members, and even health care providers hold stigmatizing views of addicts. Most these groups think that people with SUD are not trustworthy, tend to be aggressive, and tend to be criminal.\textsuperscript{39} Even drug users themselves stigmatize the route of drug administration, with intravenous drug users being considered dirtier and worse off than those who abuse oral pain medications.\textsuperscript{40}

The stigma that health care providers feel toward people with SUD is well-documented. When physicians hold beliefs about the causes of addiction that are stigmatizing and moralizing, this creates significant barriers to people with SUD obtaining adequate treatment.\textsuperscript{41} Not only are

\textsuperscript{35} Anne Marie Lavack, \textit{Using social marketing to de-stigmatize addictions: A review}, 15 \textit{Addiction in Research and Theory}, 479, *3 (2007)
\textsuperscript{36} Anne Marie Lavack, \textit{Using social marketing to de-stigmatize addictions: A review}, 15 \textit{Addiction in Research and Theory}, 479, *3 (2007)
\textsuperscript{40} Peter Flom, et al., \textit{Stigmatized drug use, sexual partner concurrency, and other sex risk network and behavior characteristics of 18 to 24-year-old youth in a high-risk neighborhood}, 28 \textit{Sexually Transmitted Diseases}, 598 (2001)
\textsuperscript{41} Katharine Press, et al., \textit{What Patients with Addiction Disorders Need from their Primary Care Physicians: a qualitative study}, 32 \textit{Substance Abuse} 349-355 (2016); Moira Ray, et al., \textit{Patient and Provider Comfort Discussing Substance Abuse}, 45 \textit{Family Medicine} 109 (2013); see also Leonieke
patients less likely to seek care because they feel judged or ashamed, these physicians might ignore addiction risk factors due to personal discomfort with the disorder. Additionally, patients will not be completely honest with their providers when they perceive these moralized and judgmental beliefs. This will lead to under-treatment or inappropriate treatments.

b. Structural Stigma

Structural stigma is manifest by public and private actors, including judges, prosecutors, legislators, social services, banks, insurance companies, restaurants, schools, and clubs. Stigma at the structural level appears as an endorsement of discrimination, which contributes to public and self-stigma. Structural stigma places unfair limits on someone’s exercising their civil rights due to the label of addict, rather than being based on any observable behavior. Examples include “discriminatory legislation that places restrictions on jury service, voting, holding political office, and parental custody rights” as well as discriminatory hiring or admissions policies based on stereotypes. It also includes the lack of parity between insurance coverage for addiction treatment and other forms of medical treatment. It remains too soon to tell whether federal parity is helping to increase treatment and reduce stigma, but there continue to be many


Lily Frank and Saskia Nagel, Addiction and Moralization: the role of the underlying model of addiction, 10 NEUROETHICS 129, 133 (2017)


Colleen Barry, Howard Goldman, Haiden Huskamp, Federal Parity In The Evolving Mental Health And Addiction Care Landscape, 35 HEALTH AFFAIRS, 1009, 1015 (2016) (“[T]he incentives for health plans to avoid adverse selection do not go away in the presence of federal parity, since there will still be variation across plans with respect to the generosity of the mental health and substance use disorder benefits offered. The next five years will be critical to gaining a detailed picture...”)
aspects of the delivery of addiction treatment that are less accessible and inferior to regular medical care.\textsuperscript{48}

Stigma has far-reaching effects, and impacts how people think about addressing SUD as a social problem. For example, 43% of respondents say they are opposed to individuals with drug addiction receiving equivalent insurance benefits, with 49% opposed to increased government spending on treatment and a whopping 76% opposed to increased government spending on housing.\textsuperscript{49} These percentages are much higher than those for mental illness generally, and have been tied to stigma.

The fact that people with SUD are overrepresented in the criminal justice system, is both a consequence and a source of structural stigma.\textsuperscript{50} In a large study of attitudes toward people with OUD, higher levels of stigma were associated with greater support for punitive policies.\textsuperscript{51} These punitive policies included greater criminalization, and permission to discriminate in housing and employment. Those who held greater stigmatized views of SUD also had lower support for public health-oriented policies like demanding insurance parity between physical and mental health services, and improving treatment access and harm reduction strategies.\textsuperscript{52}

c. Self-Stigma

Self-stigma includes negative views that stigmatized individuals believe others think about them. It also refers to negative thoughts and shame that emerge from identification with a stigmatized group.\textsuperscript{53} At the personal level, most, but not all, individuals with SUD feel considerable shame for their disorder. This is probably due in part to a feeling that they are failing in exercising agency, and in “letting ourselves down we typically let down others who rely on us.”\textsuperscript{54} Despite this internally focused shame,
there is certainly a component that is also caused by the external, social stigma that people with SUD experience.\textsuperscript{55} Perceptions of public stigma feed into normative self-concepts, so that people with SUD tend to have reduced self-esteem\textsuperscript{56}. The common representation of addicts as unreliable or untrustworthy, for example, can make affected individuals feel excluded from the public sphere, and lead them to see themselves as deserving of this shunning treatment.\textsuperscript{57} They then might withdraw from society, and stop seeking employment and participating in their communities in healthy ways. And most importantly, they will then be motivated to continue to consume drugs or alcohol to reduce the negative feelings that stem from their shame. This is what some researchers have dubbed the “looping effect,” as the label of “addict” can feed back into negative behavior, that then reinforces the negative judgments around the label.\textsuperscript{58}

It is possible to separate shame from blame. Many of us experience shame for things that are not socially blameworthy or immoral (such as having a physical disability). This is particularly acute if the stigmatized condition makes others worried that we are contagious or they experience disgust at the sight of us. But the truth is that when personal shame is experienced as part of social stigma for a behavior that is viewed rightly or wrongly as immoral, then the emotion of shame immediately stimulates feelings of moral failure.\textsuperscript{59}

Research on self-stigma in OUD has demonstrated that the higher the self-stigma, the higher the rates of depression for affected individuals.\textsuperscript{60} Additionally, people who reported recent injection drug use have been found to have significantly higher mean scores on validated measures of

\textsuperscript{55} Steve Matthews, et al., \textit{Stigma and Self-Stigma in Addiction}, 14 BIOETHICAL INQUIRY 275, 275 (2017);
\textsuperscript{56} Steve Matthews, et al., \textit{Stigma and Self-Stigma in Addiction}, 14 BIOETHICAL INQUIRY 275, 276 (2017);
\textsuperscript{57} Steve Matthews, et al., \textit{Stigma and Self-Stigma in Addiction}, 14 BIOETHICAL INQUIRY 275, 276 (2017);
\textsuperscript{58} Steve Matthews, et al., \textit{Stigma and Self-Stigma in Addiction}, 14 BIOETHICAL INQUIRY 275, 278 (2017);
\textsuperscript{59} Steve Matthews, et al., \textit{Stigma and Self-Stigma in Addiction}, 14 BIOETHICAL INQUIRY 275, 276 (2017);
\textsuperscript{60}Nikki Bozinoff, et al, \textit{Correlates of Stigma Severity Among Persons Seeking Opioid Detoxification}, 12 J ADDICT MED. 19–23 (2018); “General Self-Stigma Subscale scores were associated positively and significantly with PHQ-2 depression ($r = 0.36$, $p < .001$) as were Treatment Stigma Subscale scores ($r = 0.14$, $p = 0.004$).”
And individuals who reported having accessed detoxification care had higher rates of self-stigma than those who had not, which the researchers believed was due to a feeling that their treatments had failed. Interestingly, this same study found that the self-stigma ratings were higher for wealthier people with higher levels of education. Perhaps within this group they feel like they have more social value to lose, and are therefore more afraid of adopting the label of addict, or acknowledging their substance use disorder.

People with SUD exhibit stigmatized views toward people with more “severe” forms of the disorder. For example, one study found that people abusing pain meds “held stigmatising attitudes towards those who used heroin, with employment, education and appearance listed as reasons why people who used codeine were more ‘respectable’”. Another qualitative study of people in treatment for over-the-counter codeine dependence in Australia found there were perceptions that MAT was for “drug users”, and was for “other people”, namely those using intravenous heroin. Even among people with drug addiction, there is a hierarchy of shame and othering of those more severely affected.

2. Stigma Leads to No Treatment and Poor Treatment

Public health messaging from the last several decades has focused on instilling fear in children—painting a terrible picture of addicts as dirty, pathetic, dishonest, and homeless. While these campaigns may have worked in the past, they are now backfiring. The popular construction of the addict as a dangerous, unpredictable criminal has led to massive under-treatment. While more than 2.3 million people in the U.S. have an opioid use disorder, less than 40% receive evidence-based treatment. The reason in part, lies

64 Sasha Cooper, et al., Perceived stigma and social support in treatment for pharmaceutical opioid dependence, 37 DRUG & ALCOHOL REVIEW 262, 263 (2018)
65 Suzanne Nielsen, et al., Pharmaceutical opioid analgesic and heroin dependence: How do treatment-seeking clients differ in Australia?, 30 DRUG AND ALCOHOL REVIEW 291, 297 (2011) (“There may be societal elements at play, such as the reported perception that opioid substitution treatments—of which the majority of participants in this study were recruited from—are primarily for ‘drug users’, specifically heroin users.”)
with stigma. Stigma is routinely among the most common reasons people with SUD give for not initiating treatment or maintaining treatments that allow for sustained abstinence. Stigma has been labeled “the most important obstacle to the provision of mental health care.” As such, the U.S. Substance Abuse and Mental Health Services Administration (“SAMHSA”) prioritizes reducing stigma, as it is an essential barrier to treatment and public health goals. Stigma discourages affected individual from seeking treatment, as they do not want to adopt the label of an “addict.”

Stigma also increases shame which might perpetuate substance use, discourages their health care providers from treating them adequately, and makes insurance coverage less accessible and more expensive. Even if someone can overcome the many hurdles to receiving adequate addiction treatment, stigma is still associated with negative mental and physical health consequences.

Not only does stigma provide a reason for denying the disorder and not accessing treatment, but it also impacts the quality of the treatment that people with SUD receive. A 2015 public opinion survey found that only

67 Sara Wallhed Finn, Ann-Sofie Bakshi & Sven Andreasson, Alcohol consumption, dependence, and treatment barriers: perceptions among nontreatment seekers with alcohol dependence, 49 SUBSTANCE USE MISUSE, 762, 762 (2014); Charlotte Probst, et al., Alcohol use disorder severity and reported reasons not to seek treatment: a cross-sectional study in European primary care practices, 10 Substance Abuse Treatment, Prevention, and Policy (2015) (“Of 1,008 patients diagnosed with an alcohol use disorder (via general practitioner or patient interview) in the past 12 months, the majority (N = 810) did not receive treatment and 251 of those gave a reason for not seeking treatment. The most frequent reason was ‘lack of problem awareness’ (55.3 % of those who responded), the second most common response was ‘stigma or shame’ (28.6 %), followed by ‘encounter barriers’ (22.8 %) and ‘cope alone’ (20.9 %”).

68 NORMAN SARTORIUS, FIGHTING STIGMA: THEORY AND PRACTICE, 1 World Psychiatry 26, 27 (2002)

69 Patrick Corrigan & Katherine Niewegowski, Stigma and the public health agenda for the opioid crisis in America 59 Int. J. Drug Policy 44, 44 (2018)


71 [cite]


73 “Historically, insurers have not funded addiction treatment as generously as other costly medical services like cardiac care or organ transplant. Addicted patients were sometimes excluded from enrollment in insurance or charged very high premiums. Some insurers also opted not to cover addiction services, or pushed the costs of these services back onto patients through very high co-pays.” See, Valarie K. Blake, Engaging Health Insurers in the War on Prescription Painkillers, 11 HARV. L. & POL’Y REV. 485, 501 (2017)

19% of Americans surveyed thought medication assisted treatment (MAT)—the gold standard for opioid use disorder treatment—was the best way to treat heroin dependence, preferring strategies like Narcotics Anonymous that involve being “drug free.” At least 2.3 million people in the U.S. have OUD, yet over 60% do not receive evidence-based treatment. Only 4% of licensed MDs are approved to prescribe buprenorphine, an effective drug that helps curb cravings for opioids and withdrawal symptoms. Of over 14,000 drug treatment programs in the US, some funded by federal block grants to states, most are not staffed with a single licensed medical practitioner. If addiction were instead conceived of as a medical disease, would we see these abysmal levels of evidence-based treatment?

Take for example, provider’s feelings toward Naloxone. Naloxone is a short-acting mu-opioid antagonist that can be injected by emergency responders who encounter someone who has recently overdosed. It quickly reverses the acute effects of a drug overdose, such as respiratory depression. Despite its ability to save lives, research suggests that providers have generally negative attitudes about its use. The reasons for these attitudes include concerns about “promoting or condoning substance use,” the unsafe disposal of needles, and their feelings of frustration, futility, and powerlessness. The latter is likely due to the drug’s ability to stop this overdose, while doing nothing to prevent the next one. Buprenorphine and methadone are the drugs that helps with that problem, but they are also woefully under-prescribed.

Unfortunately, some providers and the public view MAT as problematic, as you are “substituting one opioid for another.” Part of the trouble may be with the mixed message behind the label itself, as “Medication-Assisted Treatment” communicates that 1) medication is not


76 Nikki Bozinoff, et al, *Correlates of Stigma Severity Among Persons Seeking Opioid Detoxification*, 12 J Addict Med. 19–23 (2018): (“There are efficacious treatments for opioid use disorder (OUD), however according to the National Survey on Drug Use and Health, of the 2.5 million Americans 12 years and older who misused or were dependent on opioids in 2012, fewer than 1 million received treatment with methadone, buprenorphine/naloxone or naltrexone.”)


“In 2017, former Health and Human Services Secretary Tom Price was quoted as saying ‘If we’re just substituting one opioid for another, we’re not moving the dial much.’”
the primary treatment for SUD, 2) another, unnamed treatment is. Consider, the implications for example, of referring to an insulin prescription for diabetes as “Medication-Assisted Treatment.” This label of course begs the question—is there any other treatment that is not medical? Opioid agonists such as methadone or buprenorphine, two common forms of MAT, are effective at reducing drug relapse, as they can mitigate painful withdrawal and cravings that might fuel the addiction cycle. They do so by releasing a sustained and small dose of opioids. Without MAT, the immediate withdrawal symptoms, such as sweating, shaking, and diarrhea, may resolve within a few days. Other symptoms, however, such as dysphoria, insomnia, and anxiety can linger for months, and drive drug use to self-medicate the withdrawal. These drugs literally save lives. They should be prioritized for what they are—effective medical treatments.

For individuals with OUD, more than 80% return to drug use if treated with only behavioral interventions, like Narcotics-Anonymous or psychotherapy. In contrast, treatment with adequately dosed MAT leads to only 15% of those treated continuing to use illicit opioids. Buprenorphine, in particular, “has demonstrated effectiveness in increasing treatment retention, reducing opioid use, reducing mortality, and reducing the transmission of HIV and hepatitis C,” and it has some cost, efficacy, and administering advantages over methadone, especially when combined with Naloxone. Because buprenorphine can be delivered in non-specialty settings sublingually, or via injection or implant, it can be “less stigmatizing for patients, better integrated with other medical care, maintained under a

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84 Valerie Hewell, Angel Vasquez, & Inna Rivkin, Systemic and individual factors in the buprenorphine treatment-seeking process: a qualitative study, 12 Substance Abuse Treatment, Prevention, and Policy 1, 4-5 (2017) (As one person with OUD commented: “(Withdrawal) was physical, mental, emotional, spiritual. I was a disaster for like…I think I made it…four days, and then I went back.” Participants reported withdrawal made it challenging to quit or stay off opioids without support. As one participant noted, “So I had to come off of it cold turkey, and it was a terrible, terrible experience, so I just went back to heroin.” As such, participants described MAT as being helpful in decreasing withdrawal symptoms, which allowed it to be used as a ‘stepping stone’ to recovery)
85 Christopher Evans and Catherine Cahill, Neurobiology of Opioid Dependence in Creating Addiction Vulnerability, 5 F1000 Research 1748, 1748 (2016)
86 Christopher Evans and Catherine Cahill, Neurobiology of Opioid Dependence in Creating Addiction Vulnerability, 5 F1000 Research 1748, 1748 (2016)
long-term primary care–patient relationship, and available to special populations” such as people in prison or on parole. Despite its demonstrated clinical effectiveness, because buprenorphine contains small amounts of opioids, many physicians and the public stigmatize its use, even calling into question whether someone treated with physician-prescribed buprenorphine can be considered in recovery, or sober.91

This is a dangerous perspective. Stigma surrounding MAT, and “the belief that people on MAT were still addicts and not in the recovery process,” has interfered with participants’ treatment and recovery.92 Why is MAT not respected? Because addiction is not seen as a disease. We treat lung cancer with chemotherapy toxins and radiation, realizing that these dangerous treatments would not be prescribed to someone without cancer. We should treat MAT in the same way we treat other treatment options, by prescribing it when its clinical benefits outweigh its risks. In many cases, when confronted with continued intravenous drug use and risk of overdose, the risks are easily justified.

Recognizing the barriers to accessing MAT, Congress passed the Drug Addiction Treatment Act in 2000 (“DATA 2000”), allowing physicians to request a waiver from the Controlled Substances Act requirements to treat OUD outside of a federally-regulated Opioid Treatment Program (“OTP”).93 Even still, however, fewer than 4% of licensed physicians are approved to prescribe buprenorphine.94 It is

91 One patient in recovery through MAT explained the stigma toward buprenorphine this way: “I hear it all the time: ‘you’re not sober’. And it really hurts my feelings because I worked hard...from where I was to where I (am) now...I got my own place, I got my disability, I got everything on track. And all she said was, ‘you’re still not sober.’” Valerie Hewell, Angel Vasquez, & Inna Rivkin, Systemic and individual factors in the buprenorphine treatment-seeking process: a qualitative study, 12 Substance Abuse Treatment, Prevention, and Policy 1, 4-5 (2017) see also, Barbara Andraka-Christou, America Needs the Treat Act: Expanding Access to Effective Medication for Treating Addiction, 26 Health Matrix 309, 339–40 (2016) (“The War on Drugs contributes to the underuse of buprenorphine in two ways: by stigmatizing drug-dependent individuals and by causing them to hide their illness (rather than seeking treatment) out of fear of punishment for drug possession.”)
92 Valerie Hewell, Angel Vasquez, & Inna Rivkin, Systemic and individual factors in the buprenorphine treatment-seeking process: a qualitative study, 12 Substance Abuse Treatment, Prevention, and Policy 1, 4-5 (2017)
93 The Drug Addiction Treatment Act, was passed as part of the Children’s Health Act, codified at 21 U.S.C.A. § 823 (West 2019)
94 “Surprisingly, utilization of buprenorphine is very low in the U.S., partly due to restrictions placed on prescribers under the Drug Addiction Treatment Act (DATA) of 2000. In 2013, Senator Marky introduced the Recovery Enhancement for Addiction Treatment Act (TREAT Act) in the Senate, which would loosen DATA’s patient limit restrictions and expand prescribing ability to nurse practitioners and physician assistants. Even though the bill was strongly supported by the American Medical Association, American Society of Addiction Medicine, and other professional organizations, it received scant media or public attention.” See, Barbara Andraka-Christou, America Needs the Treat Act: Expanding Access to Effective Medication for Treating Addiction, 26 Health Matrix 309, 317 (2016)
estimated that only half of the physicians with waivers actually prescribe buprenorphine, and most of those prescribe far below their capacity. Almost half of our counties in the U.S.A. lack a buprenorphine-waivered physician, and there is a significant gap between treatment need and capacity. This is despite its “high potential” to treat affected individuals, due to “its approval for use in non-specialty outpatient settings, effectiveness at promoting abstinence, and cost effectiveness.” While it can be hard to frame this concept for physicians, and may be under-reported, “many physicians explicitly cite [stigma toward patients with SUD] as a barrier.”

It is hard to imagine another disease for which there exists such a skimpy infrastructure for treatment. Visualize the public outcry, if nearly half of our counties did not have a chemotherapy or dialysis clinic, and over 60% of individuals with cancer did not get evidence-based treatment. Yet this is where we are with SUD. And it is precisely because it is seen as a moral failing rather than as a medical disease.

II. LEGAL RESPONSE TO STIGMA

A. Criminalization Fosters Stigma

To answer, “how did we get here?” we must look not just to the media depictions of addicts, but to the over-criminalization of addiction. Stigma permits criminalization, but it also is exacerbated by it. The policy feedback literature suggests that “enactment of public policies can lead to changes in public perceptions of the worthiness of the population targeted by the policy and shift political power by creating new constituencies.” Not only are policies impacted by social stigma, as politicians will rarely expend the capital to protect heavily stigmatized groups, but therapeutic and compassionate policies can reduce stigma. Criminalizing drug possession does the opposite.

Recent research demonstrates that the extent to which people stigmatize drug addiction predicts their support for punitive policies. For example, when asked whether respondents supported arresting and

95 Lloyd I. Sederer and Leslie A. Marino, Ending the Opioid Epidemic by Changing the Culture, 89 PSYCHIATRIC QUARTERLY 891, 892 (2018)
96 Rebecca Haffajee, et al., Policy Pathways to Address Provider Workforce Barriers to Buprenorphine Treatment, 54 AM. J. PREV. MED. S230 (2018)
97 Rebecca Haffajee, et al., Policy Pathways to Address Provider Workforce Barriers to Buprenorphine Treatment, 54 AM. J. PREV. MED. S230, S233 (2018)
prosecuting people who “doctor shop” to obtain multiple opioid prescriptions, roughly 17% of the variance in support for this policy could be explained by ratings of addiction stigma.\textsuperscript{99} When researchers asked whether respondents supported requiring Medicaid enrollees, suspected of “problematic” opioid use to use a single prescriber and pharmacy, again 17% of the variance in support could be explained by stigma.\textsuperscript{100} Stigma was so powerful, it shockingly explained more of the support for punitive policies than political affiliation.\textsuperscript{101} These findings provide powerful support for the idea that “reducing stigma toward individuals with prescription OUD might be one way to discourage adoption of punitive policies.”\textsuperscript{102}

Of course, the criminalization of drug use has led to greater stigma for affected individuals as well. The modern “War on Drugs” can be traced back to President Richard Nixon, as he declared in 1971 that drug abuse was “public enemy number one in the United States.”\textsuperscript{103} Seizing on this perception, Congress began passing “tough on crime” laws that criminalized use and possession of drugs, with strict mandatory minimum sentences. This continued through the presidencies of Ronald Reagan and George H.W. Bush.\textsuperscript{104}

To build support for his “War,” the Reagan administration sought to publicize the threat of crack cocaine. According to Michelle Alexander “[a]lmost overnight, the media was saturated with images of black ‘crack whores,’ ‘crack dealers,’ and ‘crack babies’—images that seemed to confirm the worst racial stereotypes about impoverished inner-city residents.”\textsuperscript{105} In 1986, in response to news coverage that suggested NBA-recruit Len Bias had overdosed on crack cocaine, Congress adopted the 1986 Anti-Drug Abuse Act, with little debate and zero hearings.\textsuperscript{106} The Act created a mandatory minimum sentence of five years for possessing five

\textsuperscript{104} Shima Baradaran, Drugs and Violence, 88 S. CAL. L. REV. 227, 249 (2015)
\textsuperscript{106} Doris Marie Provine, Race and Inequality in the War on Drugs, 7 ANN. REV. L. & SOC. SCI. 41, 45–46 (2011)
grams of crack with intent to sell, and a minimum of ten years of imprisonment for ten grams. In 1988, Congress added a five-year minimum sentence for simple possession of 5 grams of a mixture of crack cocaine. It is impossible to speak of the War on Drugs without acknowledging how disproportionately it affected people of color. Whereas powder cocaine, “associated with a wealthier, whiter class of drug users,” required possession of 500 grams to trigger a 5-year prison term, one only needed to possess a mere 5 grams of the chemically identical crack cocaine, “regarded as a drug of the black urban ghetto,” to trigger the same sentence. The disparities in sentencing were exquisitely felt by the many black communities that were devastated by these harsh penalties. These harsh penalties received public support, as they fell on the disempowered and “racial other,” which in turn led to greater dehumanization of people of color. The result, of course, is that in modern America, if you were to gaze your eyes on the criminal justice system, you would think that drug use and addiction were largely problems for the urban, poor, African-American community. Drug addicts were the “other,” and they were dangerous. Of course, we know now how biased this snapshot was. It did not capture the many white and wealthy Americans who were similarly addicted, and it did not fully capture the many people with SUD who were neither dangerous nor involved with the criminal justice system.

The present opioid addiction crisis reveals just how racialized our political responses can be. While it is laudable that legislators are now proposing bills that encourage treatment and de-emphasize criminalization, it is quite illuminating that these compassionate responses to drug addiction have only now been proposed. Perhaps because those affected by OUD are more likely to be white, middle-class, older, and living in the suburbs, there has been less “othering” of people with OUD. As we might expect, the policy and criminal justice responses to OUD do appear to be less punitive and more therapeutic. Evidence of this can be found in the Opioid Crisis Response Act, a rare, bipartisan appropriation bill passed by Congress in 2018, with 99 Senators supporting the bill. The Act provides modest funding for research into addiction stigma, and increases housing options

110 Doris Marie Provine, Race and Inequality in the War on Drugs, 7 Ann. Rev. L. & Soc. Sci. 41, 42 (2011)
for people in recovery. Additionally, several federal agencies have responded to the opioid crisis with a much more therapeutic and prevention-based approach. However, there remains a dire need for more funding of addiction treatment programs and scaling-up of physicians authorized to provide MAT.

The Department of Health and Human Services ("HHS") developed a five-point evidence-based strategy in 2017. The five points are: (1) increasing addicts’ access to recovery and treatment services; (2) improving access to medications that reverse overdoses; (3) improving data collection; releasing data more promptly to improve public health response; (4) research into pain and addiction, including development of new treatments; (5) reducing inappropriate use of opioids by developing better evidence-based pain treatment. This effort is laudable, and does exactly what should be done: treating addiction as a disease with devastating public health impacts. We certainly saw no such political response to the Crack Cocaine epidemic of the 1980s and 1990s, which dehumanized addicts and criminalized addiction.

Despite the greater efforts to respond to OUD through drug courts and harm reduction programs, addiction remains heavily criminalized.

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112 Sec. 404 of the Opioid Crisis Response Act states, "Building communities of recovery: This section awards grants to recovery community organizations, five million dollars each fiscal year from 2019-2023. Recovery community organizations are independent non-profit organizations that mobilize resources and are governed by people in recovery for substance abuse disorders. The funds may be used to build connections between recovery networks and other recovery support services, reduce the stigma associated with substance use disorders, and conduct outreach on issues relating to substance use disorders and recovery. The Secretary shall give special consideration in awarding these grants to rural areas."

113 Given the high rates of opioid addiction in the Medicare population, the Centers for Medicare and Medicaid Services ("CMS") now require much more oversight when Medicare beneficiaries are prescribed opioids. All Medicare Part D plan sponsors are "required to have a written plan to reduce overuse of opioids, using tools such as case management, coordinated care among beneficiaries’ doctors, better management of the plan’s formulary, and safety edits when patients fill prescriptions." See, Dana Schilling, Senior Citizens and the Opioid Crisis, 328 ELDER LAW ADVISORY NL 1 (2018). Additionally, recognizing the systemic factors that can drive addiction, the Food and Drug Administration (FDA) now requires prescriber education for opioids. Nearly half of the states have received Center for Disease Control ("CDC") grants to "prevent, deal with, and track overdoses," and 44 states obtained CDC grants to fund prescription drug monitoring programs (PDMPs). See, id.


115 Testimony of Giroir and Brandt, Senate Committee on Finance Hearing: Tackling Opioid and Substance Use Disorders in Medicare, Medicaid, and Human Services Programs, available at https://www.finance.senate.gov/download/04192018-joint-testimony, at p. 3, see also, Dana Schilling, Senior Citizens and the Opioid Crisis, 328 ELDER LAW ADVISORY NL 1 (2018).

116 Barbara Andraka-Christou, What Is "Treatment" for Opioid Addiction in Problem-Solving Courts? A Study of 20 Indiana Drug and Veterans Courts, 13 STAN. J. CIV. RTS. & CIV. LIBERTIES 189, 191 (2017) ("Unlike regular courts whose primary duty it is to arbitrate civil and criminal issues, problem-solving courts focus on solving underlying problems of communities through the rehabilitation of offenders in the criminal justice system. Drug courts are one type of problem-solving court...")
According to the Federal Bureau of Investigation statistics, in 2016 alone, 1.57 million drug arrests were made in the United States.\(^{118}\) That is one drug arrest every 20 seconds, and represents more than three times the arrests made for all violent crimes combined.\(^{119}\) In 2015, the overwhelming majority of drug arrests, some 84\%, were for possession only, and did not include distribution or sales of drugs.\(^{120}\)

The stigma from incarceration itself can lead to a “why try?” effect, where people anticipate stigma and thus see no point in trying to integrate back into their communities.\(^{121}\) Conviction can also reduce access to other, derivative rights. Having a criminal record tied to drug use can negatively impact child custody, voting rights, employment, business loans, licensing, student aid, and even public housing.\(^{122}\) While it is not unfair discrimination to deny someone custody of their child when they are too dependent on opioids to safely take care for their kids, the concern here is that the label of “addict” will be doing too much punitive work. Rather than evaluating the antisocial behavior of that individual, the label of “addict” will alone persuade judges to deny parents custody in ways that might not be in the child’s best interests.

To be sure, criminalization of addiction would be better justified, even given the stigma it creates, if it worked to deter drug use. But based on data compiled by the Pew Charitable Trust, imprisonment for drug crimes does not reduce drug use, arrests, or overdose deaths.\(^{123}\) By any measure that matters, criminalization is not working.\(^{124}\) In 2000, Portugal decriminalized drug use and replaced criminal sanctions for those who possessed more than a small amount with civil penalties and public health


\(^{122}\) Sources: U.S. Census Bureau; Bureau of Justice Statistics; and http://www.drugpolicy.org/resource/drug-war-mass-incarceration-and-race-englishspanish


interventions. Dr. João Goulão, Portugal’s national drug policy director who led the reform said, “the biggest effect has been to allow the stigma of drug addiction to fall, to let people speak clearly and to pursue professional help without fear.” The country has seen a steady decline in the rate of new HIV infections as well as overdose deaths.

B. Anti-Discrimination Statutes Cannot Effectively Mitigate Stigma

Anti-discrimination statutes prohibit discrimination based on someone’s identity or observable characteristics. We therefore have federal statutes that prohibit discrimination in housing, employment, and public accommodations, based upon specific protected statuses, such as race, religion, sex, disability status, or genetic mutations. Another way of thinking about this is to require that people judge someone based on the content of their actions rather than on their belonging to a particular group, which is typically stigmatized. To demonstrate how the anti-discrimination statutes work in the context of addiction, let’s analyze the Americans with Disabilities Act (“ADA”).

Recognizing that people with physical or mental disabilities have rights to fully participate in all aspects of society, Congress passed the ADA to prohibit discrimination in employment and public accommodation. It was later amended in 2008. The ADA prohibits companies with more than fifteen employees from discriminating against a qualified individual on the basis of that person's disability, or perceived disability. Addiction qualifies as a disability, if it physically or mentally impairs the employee and limits the employee in a major life activity, such as learning or taking care of oneself. Once an employer is aware of an ADA-defined disability, she must then make “reasonable accommodations to the known physical or mental limitations” of the individual.

125 Hannah Laqueur, Uses and Abuses of Drug Decriminalization in Portugal, 40 LAW & SOC. INQUIRY 746, 747 (2015) (“The law did not alter the criminal penalty prohibiting the production, distribution, and sale of drugs, nor did it permit and regulate use. Rather, Portugal decriminalized drug use, which, as defined by the European Monitoring Centre for Drugs and Drug Addiction (EMCDDA), entailed the removal of all criminal penalties' from acts relating to drug demand: acts of acquisition, possession, and consumption.”)
126 NIGEL HAWKES, HIGH AND LOWS OF DRUG DECRIMINALISATION, 343 British Medical Journal 1, 1 (2011)
129 See 42 U.S.C.A. § 12102 (West 2019)
130 See, 42 U.S.C.A. § 12102 (West 2019)
There are a few ways that the anti-discrimination statutes, such as the ADA, will not be enough to protect people with SUD from illegal discrimination. In theory, addiction can meet the definition of a disability, and therefore must be accommodated so long as the accommodation does not create an “undue burden” for the employer. But in practice, it is easy to fire people with SUD, or exclude them from broad classes of employment. First, if you are ever intoxicated at work, your behavior is understandably grounds for discipline and is not protected by the ADA. Even so, the employer must apply any disciplinary policies equally to all affected employees. If your intoxicated behavior embarrassed the company, even if it occurs outside of your employment, you may be fired and it will not violate the ADA. There are often pretextual reasons to terminate someone with an addiction. If you are covering your addiction well, you will not be protected under the ADA, as the employer needs to regard you as having a disability. It is often difficult to prove that the employer knew of your addiction unless you exhibit intoxicated behavior, in which case you are also not protected. If you are recently using, you are not a qualified person with a disability, and are not protected under the ADA or the Family Medical Leave Act, even if none of your behavior led your employer to believe you were impaired. If you disclose your SUD to seek unpaid time off for treatment, employers can then fire you and claim it is due to a business necessity reason, even if they had no idea you had any problem before you alerted them. If someone is participating in an addiction treatment program, has successfully completed a treatment program, or is no longer using illegal drugs, they are not excluded as a “qualified individual.” However, once an employer knows about an employee’s SUD, the “business necessity” exception provides for far-reaching exclusions to protection, even for people who are now sober. Further, if you are now sober, some courts will hold that you are no longer

132Renaud v. Wyoming Department of Family Services, 203 F.3d 723 (2000); see also
134See Maddox v. University of Tennessee, 62 F.3d 843 (6th Cir. 1995); see also Labrucherie v. Regents of the Univ. of California, U.S. app. LEXIS 17755 (9th Cir. 1997)
136See Zenor v. El Paso Healthcare System, 176 F.3d 847 (5th Cir. 1999) See also, 42 U.S.C.A § 12114 (West 2019)(“For purposes of this subchapter, a qualified individual with a disability shall not include any employee or applicant who is currently engaging in the illegal use of drugs, when the covered entity acts on the basis of such use.”)
13742 U.S.C.A. § 12114 (West, 2019)
13842 U.S.C.A. § 12113 (West, 2019); See also, EEOC v. Exxon Corp. 967 F. Supp. 208 (N.D. Tex 1997) (Exxon successfully argued that, due to the high rates of relapse among rehabilitated substance abusers, they could exclude all of them from “designated positions,” even without performing an “individualized assessment.”)
experiencing a “disability,” nor are you “regarded as having a disability,” if the impairment to your life is not substantial enough.\textsuperscript{139} This of course confuses the status of someone in recovery, as “in this context, [sobriety] is not synonymous with a cure; it is a personal process of movement toward a meaningful, purposeful, and satisfying life.”\textsuperscript{140} Despite the stigma attached to current or previous SUD, employees are not fully protected from discrimination while they are keeping their behavior under control, while they are using, while they are exhibiting behavior of intoxication, or when they are in recovery. This leaves very little protection for people with SUD. Given the problems with addiction being a heavily stigmatized disorder based on biology and behavior, it does not fit neatly within the classes protected under various anti-discrimination statutes. As illustrated in its ineffective protection under the ADA, it is no wonder that people with SUD are regularly discriminated against.

In addition to stigma toward SUD, its treatment is stigmatized too. The Legal Action Center in New York reports that nursing homes are discriminating against potential elderly residents who use MAT (such as methadone or buprenorphine), and will deny them residency based upon their MAT use. Some organ transplant patients cannot be listed if taking MAT,\textsuperscript{141} and in other instances MAT patients have been forced to taper to maintain custody or their jobs. This sort of discrimination for receiving medical treatment, is not protected by the ADA or other federal anti-discrimination statutes. State anti-discrimination statutes might provide greater, but still spotty, protection.

III. \textbf{THE MORAL CHOICE MODEL MUST BE REPLACED WITH THE IDM}

\textit{A. The Competing Causal Models of Addiction}

A well-accepted view of SUD is that it unfolds as a three-step process. First comes the binge or intoxication episodes. After enough of these episodes, some individuals will become addicted (either through weak character, as some posit, or through genetic vulnerability to physical dependence, as I suggest). Once the drug is metabolized there is a hangover, which leads to negative affect and withdrawal symptoms. This in turn leads to the third step of preoccupation, where desire for the drug intensifies.

\begin{footnotesize}
\begin{enumerate}
\item Johnson v. NYC Office of Alcohol, LEXIS 41986 (S.D. N.Y. 2018)
\item National Academies of Science, \textit{Ending Discrimination Against People with Mental and Substance Use Disorders: The Evidence for Stigma Change}, available for download at http://nap.edu/23442, at p. 1
\item Woods & Joseph, Stigma from the Viewpoint of the Patient. 34 J. Addictive Dis 247 (2015)
\end{enumerate}
\end{footnotesize}
and prompts the next intoxication episode. This three-step process describes the cycle of drug use, whether you think of this process in terms of a learning, memory, developmental, reward, impulse, or chemical or neuroinflammatory disorder. Stressful life events can lead to continued drug use to self-medicate and escape the stress or pain.

How we conceive of addiction impacts our response. Some have argued that “whether addiction is a disease is much ado about nothing, since all parties agree it is ‘unquestionably destructive.’” But it does matter. While our current moral agency model discourages investments in treatment and encourages investment in criminalization, a model that is on equal footing with lung cancer or diabetes permits greater research funding and insurance parity. So how do we get there? We first need to dig a little deeper into the various models.

There are as many models of addiction as there are drugs to abuse. It makes sense then, that our policy response to addiction has been inconsistent and fractured. Scholars have argued that the best way of conceptualizing addiction is to see it as either a disorder of development, trauma, risk-taking, choice, associative reward learning, memory, opponent biological processes, genetics, or neuroscience. Unfortunately, many see these as dichotomous and competing, as opposed to complementary.

Most scholars keen on identifying the “correct” model of addiction fall in the mutually exclusive “brain disease” or “moral choice” camps, even though there are many other models. Philosophers and legal scholars have spilled much ink on this “oftentimes heated scholarly debate” as to whether we ought to think of addiction as a moral choice or a disease.

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142 Emmanuel Darcq and Brigitte Lina Kieffer, Opioid Receptors as “Drivers” of the Onset, Progression, and Maintenance of Addiction, 19 NATURE REVIEWS NEUROSCI 499, 504 (2018)
143 Christopher Evans and Catherine Cahill, Neurobiology of Opioid Dependence in Creating Addiction Vulnerability, 5 F1000 RESEARCH 1748, *1 (2016)
144 “[H]ow patients understand their addiction may shape their health behaviors, relationship with their care team, and willingness to accept treatment.” See Helen Jack, et al., THE AMERICAN J. OF MEDICINE e1, e1 (2018)
145 OWEN FLANAGAN, ADDICTION DOESN’T EXIST, BUT IT IS BAD FOR YOU, 10 EUROETHICS 91, 91 (2017)
146 As every single one of these processes is mediated by the brain and any deficits can be explained in terms of brain circuitry and chemistry, they are easily adopted into an explanatory model that emphasizes brain disorder. The moral choice model stands alone in terms of failing to make any explanatory space for addiction as a disorder of neurological and psychological processes. Marc Lewis, Addiction and the Brain: development, not disease 10 Neuroethics 7, 8 (2017)
147 “Long-standing debates concerning the moral status of addiction have arisen from one of two perspectives: either addiction is a disease of the brain, or addiction is a matter of weak will.” See, Daniel Buchman, Wayne Skinner, and Judy Illes, Negotiating the Relationship Between Addiction, Ethics, and Brain Science, 1 AJOB NEUROSCIENCE 36, 40 (2010)
148 Lily Frank and Saskia Nagel, Addiction and Moralization: the role of the underlying model of addiction, 10 EUROETHICS 129, 129 (2017) (He writes, that “[w]hat most don’t see because of the meager dialectical offerings – addiction is either a moral or a brain/gene disorder – is the prospect that one can see addiction as involving biographically interpretative assessment of one’s own reason
Though most neuroscientists, leading addiction researchers, and even government entities promote addiction as a chronic brain disease, a recent review shows “varied adoption of the brain disease model among addiction treatment providers, with some viewing addiction as a purely behavioral problem or maladaptive coping mechanism.” The disease model is typically associated with being less moralized, as the emphasis is not on the moral character of the individual, but rather on biological risk factors, and a brain that becomes compelled to use drugs. The choice model invites greater ascriptions of stigma, blame and personal responsibility for choosing hedonism over abstinence. The moral choice model is the one we see most often in our history and popular culture. It has done great violence to the treatment of addiction, both individually and as a public health crisis.

B. The Moral Choice Model of Addiction

Those advocating the “choice model” emphasize that people can and do stop using drugs, with sufficient incentives. Some (though not many) people with SUD achieve recovery without any medical treatment, and they may have never identified with being “sick” or now “cured.” To this camp, addiction is a failure to exercise agency or self-control, as well as a failure to “live up to the standards of a good life.” Some of the moral choice theorists posit that personal shame is a necessary condition for addiction, but that shame need not result in a “moralized” view of addiction. This interpretation strains credulity. The examples they provide, of people feeling ashamed of things that are outside their control, such as body deformities, are likely holdovers from shame that was directed at people thought to carry infection. To continue this sort of blameless shame, or suggest that addiction relies upon it, is to continue this sort of irrational holdover.
Proponents of the choice model argue that aspects of drug-taking require voluntary action, such as driving to meet a dealer, or leaving work early to shoot-up. Further, the pleasure that may be derived from satisfying a drug craving is understood by some as a rational expression of individual preference. And rather than seeing the brain changes that can be visualized in people with SUD as evidence of a brain disease, they argue that the very nature of the brain is to change. Thus, brain changes are not equivalent to disease.

While the choice model is the one that most readily lends itself to an account that blames the addict and finds them morally responsible, there are aspects of the choice model that proponents argue may benefit individuals with SUD. For example, envisioning addiction as a brain disease might take too much pressure off society to prevent its social determinants, such as housing, unstable home lives, personal safety, and employment. Further, deterministic thinking might lead people with SUD to give-up efforts at sobriety if the disease is “fixed” in their brains. Of course, this characterizes just one way that the disease model can be operationalized, and assumes that there is insufficient funding to address treatment and the public health aspects of addiction.

C. The Brain Disease Model of Addiction

In contrast to the choice model, the disease model of addiction conceptualizes it as a “severe, chronic stage of substance-abuse disorder, in which there is substantial loss of self-control, as indicated by compulsive drug-taking despite the desire to stop taking the drug.” The definition from the National Institute on Drug Abuse (NIDA) goes further to state that

158 Lily Frank and Saskia Nagel, Addiction and Moralization: the role of the underlying model of addiction, 10 NEUROETHICS 129, 130 (2017)
159 Lily Frank and Saskia Nagel, Addiction and Moralization: the role of the underlying model of addiction, 10 NEUROETHICS 129, 130 (2017)
162 Nora Volkow, et al., Neurobiological advances from the brain disease model of addiction, 374 N. ENG. J. MED. 363, 364 (2016)
addiction “is considered a brain disease because drugs change the brain—they change its structure and how it works. These brain changes can be long-lasting, and can lead to the harmful behaviors seen in people who use drugs.” While addiction no doubt changes the brain, the IDM I advocate goes further than merely demonstrating brain changes through functional or structural brain imaging. Rather, the IDM recognizes that neurobiological vulnerabilities can lead to addiction, as opposed to just flow from it. It further situates the brain inside a human being, which has been exposed to various environmental stressors and responds differently to drug use. Given the potential nuance that a disease model such as the IDM provides, it is perplexing that scholars fail to see how it can accommodate and respond to its critics.

Nonetheless, the brain disease model of addiction remains controversial. I have given a great deal of thought as to why this might be so. I believe proponents of the brain disease model have unnecessarily overplayed their hand, and painted addiction in neuro-essentialist and deterministic ways. Using metaphors like the “hijacking” of the brain, some suggest that the compulsion to use drugs is so great that you might not be legally responsible for criminal acts stemming from your addictive behaviors. But the latter does not flow from the former, because the brain is never completely hijacked.

Put simply, some advocates for the brain disease model have fallen into the same trap the moral choice camp has fallen in to, which is to think that free will and biological causes are mutually exclusive. Addiction is either a disease of the brain or it is a voluntary, moral choice. One or the other. Black and white. However, to say that addiction is a brain disease is not to say that the affected individual loses all capacity to make reasons-based decisions. Even Owen Flanagan, who argues that addiction is a failure of agency, recognizes that the dichotomy between morality and biology is false. There are aspects of addiction that suggest the person is making some constrained choice, and there are aspects of addiction that

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164 Nora Volkow and George Koob, Brain Disease Model of Addiction, why is it so controversial? 2 LANCET PSYCHIATRY 677, 677 (2015)
165 Daniel Buchman, Wayne Skinner, and Judy Illes, Negotiating the Relationship Between Addiction, Ethics, and Brain Science, 1 AJOB Neuroscience 36, 36 (2010) (“Although a brain disease model legitimates addiction as a medical condition, it promotes neuro-essentialist thinking, categorical ideas of responsibility and free choice, and undermines the complexity involved in its emergence.”)
166 Owen Flanagan, The Shame of Addiction, 4 FRONTIERS IN PSYCHIATRY 120 (2013)
follow a brain disease model. Recently, "Endorsing a [disease model of addiction]" is not "inconsistent" with a free-will model. Conversely, even if there is a voluntary choice involved in the decision to use drugs for the first, second, or third times, once someone has developed SUD, all of their “choices,” including those unrelated to drug use, do become significantly constrained, if not perfectly determined. The volitional nature of behavior is more pronounced in the initiation phase of disease, but brain adaptations can reduce volition leading to habits, altered reward processing, stress reactivity, and negative affect and physiology of withdrawal. More will be said about this infra at [x].

D. Neither Purely a Brain Disease nor Purely a Moral Choice: the IDM

In adopting the IDM, we need not rely on the “fundamental psychological error.” The disease model does not suggest that addiction is caused only, or even predominantly, by the brain. What’s more, this Article is about moral justifications for treatment, not moral justifications for punishment. While our models of addiction impact our criminal laws, it is not an inevitable step from a disease model to an argument that would necessarily be relevant to the criminal law. Neuro-genetic factors are but one of many relevant types of causes. A disease model would hopefully encourage compassion in how we prosecute drug possession, just as a history of child abuse or other psychiatric disorders might engender mercy. But that does not mean that the individual could never be legally responsible for his actions. Indeed, with increased access to effective and

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167 Rather than being a disease or a choice, addiction might be the result of psychological or neurological mechanisms that diminish reason-responsiveness, due to associative learning. For example, according to Marc Lewis, addiction is a “habit that grows and self-perpetuates relatively quickly, when we repeatedly pursue the same highly attractive goal. Or, in a phrase, motivated repetition that gives rise to deep learning.” Marc Lewis, THE BIOLOGY OF DESIRE: WHY ADDICTION IS NOT A DISEASE, New York: Public Affairs, at p. 174 (2015)


169 “Observers will continue to rate the addict as culpable long after the drug is ingested for ‘personal choice’ and functional dysregulation and structural alterations have materialized in brain areas that regulate motivation and self-regulation in the face of escalating consequences.” G.F., Koob, and M. Le Moal. What is addiction, NEUROBIOLOGY OF ADDICTION. Koob and Le Moal (eds), Elsevier/Academic Press, Amsterdam/Boston (2006): 1-22.

170 Christopher Evans and Catherine Cahill, Neurobiology of Opioid Dependence in Creating Addiction Vulnerability, 5 F1000 RESEARCH 1748, *7 (2016)

171 “[D]iscovery of genetic or of any other physical or psychosocial cause of action raises no new issues concerning responsibility, and discovery of such causes does not per se create an excusing or mitigating condition for criminal conduct or any other type of behavior.” Stephen J. Morse, Addiction, Genetics, and Criminal Responsibility, LAW & CONTEMP. PROBS., Winter/Spring 2006, at 165, 166.
cheaper SUD treatments in the future, prosecutors might have greater moral justification for prosecuting people who do not seek it.

Finally, adopting the IDM does not require that the individual with SUD be capable of being “cured.” There are many diseases for which a cure is never likely, and the best that can be hoped for is remission or sustained recovery. And treating addiction as a disease does not mean that we should ignore the social determinants of health, such as high-stress environments, just as we should not ignore the social determinants of lung cancer, diabetes, depression or AIDS. Many of the criticisms of the brain disease model rely on misunderstandings of the complex etiology of ‘disease,’ and bring to bear a very constrained model of what it means for something to be a choice, as well as what it means for something to be a disease. The two are not wholly incompatible. What can be said, uncontrovertially, is that whether one adopts a disease model, a choice model, or something else, that the mechanisms involved in addiction are neurobiological. The three phases of addiction are craving, binging, and withdrawal, and the resulting physical dependence can be understood and explained by neurobiological mechanisms. Further, the process from drug use to mild SUD, to moderate SUD, and then to severe SUD, otherwise labeled full-blown addiction, can be explained by neurobiological systems.

E. The Neurobiology of Addiction Does Not Support the Moral Choice Model

1. The Brain is Not Hijacked, but Is Constrained in Patients with SUD

The neurological processes behind the development of addiction have been extensively studied. There are different ways to explain what is happening at both the neurological and psychological levels. I will offer a few of those explanations here, all of which fit neatly within the IDM. It is well accepted that drugs activate reward regions in the brain by causing sharp increases in the release of dopamine, which sets off a cascade of

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172 Examples include lung cancer, anorexia nervosa, and substance use disorder.
174 Substance use disorder (SUD) is a diagnostic term used in the DSM-5 referring to recurrent use of alcohol or other drugs that cause clinically and functionally significant impairment. This disorder can be classified as mild, moderate, or severe. Addiction is used to indicate the most severe stage of this SUD process, in which individuals have a substantial loss of self-control, and will use the drug despite the desire to stop, and in the face of negative consequences. See, Nora Volkow, et al., Neurobiologic Advances From the Brain Disease Model of Addiction, 374 NEW ENG. J. MED. 363, 364 (2016).
reinforcement learning and Pavlovian conditioning.\textsuperscript{175} As with any other form of motivated learning, the greater the associated reward, the more work someone is willing to do to get it.\textsuperscript{176} When it comes to “natural reward,” dopamine cells reduce their firing in response to food or sex, once someone is considered sated. This is not the case with drugs, which can circumvent the satiation mechanisms of the brain.\textsuperscript{177} However, this effect tapers off. With repeated use, dopamine cells cease firing in response to the drug itself, and instead fire in anticipation of the conditioned stimuli, or drug cue.\textsuperscript{178}

Another complementary way of explaining drug use is in terms of “opponent process” theory. Under this explanatory model, once the positive euphoric state is triggered (the a-process), brain mechanisms will work to reduce the intensity of this affective state (the b-process). The intoxication phase, or the “a-process,” motivates the individual to seek more of that pleasurable stimuli—in this case drugs.\textsuperscript{179} The a-process does this by triggering dopamine and opiate peptides to bind to receptors in the VTA and nucleus accumbens, which mirrors the reinforcement learning process.\textsuperscript{180}

After the effects of an opioid wear off, the b-process begins. This b-process produces sharp declines in dopamine and opioid peptide neurons, and increases stress steroids such as adrenaline and corticotropin-releasing factor (CRF). While these operate to return an individual to baseline, in someone with SUD, the b-process fuels the addiction, by generating physical dependence and withdrawal at the synaptic level. The sharp increase in CRF and adrenaline, coupled with the desensitization of dopaminergic receptors and the release of dynorphin, leads to mood irritability, emotional pain, malaise, dysphoria, alexithymia, and as discussed above, increases the threshold for experiencing reward.\textsuperscript{181} The pathway between the paraventricular nucleus of the thalamus (PVT) and the nucleus accumbens has been identified as a prominent neural circuit in

\textsuperscript{175} Nora Volkow, et al., \textit{Neurobiologic Advances From the Brain Disease Model of Addiction}, 374 NEW ENGL. J. MED. 363, 364 (2016).
\textsuperscript{176} Nora Volkow, et al., \textit{Neurobiologic Advances From the Brain Disease Model of Addiction}, 374 NEW ENGL. J. MED. 363, 366 (2016).
\textsuperscript{177} Nora Volkow, et al., \textit{Neurobiologic Advances From the Brain Disease Model of Addiction}, 374 NEW ENGL. J. MED. 363, 366 (2016).
\textsuperscript{178} Nora Volkow, et al., \textit{Neurobiologic Advances From the Brain Disease Model of Addiction}, 374 NEW ENGL. J. MED. 363, 364 (2016).
relaying aversion and memory of withdrawal symptoms. Amazing new research, silencing this pathway through optogenetics, suppressed physiological withdrawal and aversion in the drug dependent state. This sort of methodology, that can demonstrate cause and effect by disrupting the neural circuitry of the “addicted brain,” is promising, but in need of replication.

Repeated exposure to dopamine-triggering drugs leads to adaptions in the circuitry of the brain, most notably in the striatum, ventral tegmental area, (VTA) and basal forebrain. Eventually the intensity of this process euphoria levels off in response. We can see this as each time the same drug is used, the release of dopamine is diminished in the synapses. This explains why people with addiction “chase the dragon,” and no longer experience the same euphoria they first experienced when using the drug. This eventual attenuation makes the brain’s reward system much less sensitive to stimulation of rewards of all types—including drugs but also food, relationships, and activities. The neuroscientific changes are engrained in the brain and take a long time to reverse. It is because of this altered neurobiology that the analogy of addiction to lung cancer or diabetes stops being helpful, however. Addiction is different. The disease itself can lead you to sabotage your own recovery, by creating a physiological pull to use drugs, despite strong personal desires to quit. It would be as if lung addiction had a symptom that made you averse to chemotherapy or surgery. Once someone develops the disorder of addiction, the motivation to use drugs can eclipse the motivation to eat or sleep. Avoiding withdrawal might become the chief motivating factor in one’s life.

The brains of people with severe SUD have adapted to expectations of sustained, high volume drug use, and at the same time their receptors are increasingly insensitive to the dopamine that is being produced. This helps

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182 Christopher Evans and Catherine Cahill, Neurobiology of Opioid Dependence in Creating Addiction Vulnerability, 5 F1000 RESEARCH 1748, *7 (2016)
183 Christopher Evans and Catherine Cahill, Neurobiology of Opioid Dependence in Creating Addiction Vulnerability, 5 F1000 RESEARCH 1748, *7 (2016)
184 JOSHUA JENNINGS, ET AL., DISTINCT EXTENDED AMYGDALA CIRCUITS FOR DIVERGENT MOTIVATIONAL STATES, 496 Nature 224, 224 (2013)
185 “[D]rug consumption triggers much smaller increases in dopamine levels in the presence of addiction (in both animals and humans) than in its absence (i.e., persons who have never used drugs.” See, Nora Volkow, et al., Neurobiologic Advances From the Brain Disease Model of Addiction, 374 NEW ENG. J. MED. 363, 366 (2016).
186 “[D]rug consumption triggers much smaller increases in dopamine levels in the presence of addiction (in both animals and humans) than in its absence (i.e., persons who have never used drugs.” See, Nora Volkow, et al., Neurobiologic Advances From the Brain Disease Model of Addiction, 374 NEW ENG. J. MED. 363, 366 (2016).
to explain the observed behavior of people with addiction. Neural circuit adaptations make individuals more reactive to stress.\textsuperscript{188} This in turn leads to greater vulnerability to depression and anxiety, which in turn can lead to greater drug use as a form of “self-medication” to ease the anxiety.\textsuperscript{189} Many say that they really want to stop using, but get caught in the vicious cycle of administering drugs to escape the anxiety and physical distress that is produced by their vulnerable neuro-circuitry, and physical withdrawal. And at the same time, what used to be an impulsive, or voluntary, choice to use drugs has now become much more compelled.

Trevor Robbins’ lab at Cambridge has studied this shift in neurobiology when people go from impulsive to compulsive drug use.\textsuperscript{190} He posits that individuals first associate the drug with either euphoria or just relief from aversive conditions.\textsuperscript{191} Then, when drug use is escalated, in vulnerable individuals it leads to dependence. More will be said about what makes individuals vulnerable, infra, at \([x]\). The third step to addiction, or severe SUD, comes when reward circuitry is changed, leading to insensitive sensitization and a strong motivation for drug use.\textsuperscript{192} Specifically, Robbins’ research describes:

“evidence that the switch from controlled to compulsive drug seeking represents a transition at the neural level from prefrontal cortical to striatal control over drug-seeking and drug-taking behaviours as well as a progression from ventral to more dorsal domains of the striatum, mediated by its serially interconnecting dopaminergic circuitry. These neural transitions depend upon the neuroplasticity induced by chronic self-administration of drugs in both cortical and striatal structures, including long-lasting changes that are the consequence of toxic drug effects.”\textsuperscript{193}

\textsuperscript{190} BARRY EVERITT, ET AL., \textit{NEURAL MECHANISMS UNDERLYING THE VULNERABILITY TO DEVELOP COMPULSIVE DRUG-SEEKING HABITS AND ADDICTION}, 363 Philosophical Trans. of the Royal Society 3125, 3125 (2008)
\textsuperscript{192} Christopher Evans and Catherine Cahill, \textit{Neurobiology of Opioid Dependence in Creating Addiction Vulnerability}, 5 F1000 RESEARCH 1748, *3 (2016)
\textsuperscript{193} Christopher Evans and Catherine Cahill, \textit{Neurobiology of Opioid Dependence in Creating Addiction Vulnerability}, 5 F1000 RESEARCH 1748, *3 (2016)
There is a substantial literature documenting reduced white matter and damaged myelin in the brains of people with OUD,\(^\text{194}\) as well as deficits in blood-oxygenated level responses (BOLD) evidenced on functional magnetic resonance imaging. People with SUD have “showed less activation in the frontal lobe than healthy subjects during the cocaine cue tapes, suggesting that their ability to control their cue responses was inhibited.”\(^\text{195}\) In a different study, researchers found that chronic cocaine abusers had abnormally low levels of activity in midline areas of the anterior cingulate that are crucial for cognitive and behavioral control.\(^\text{196}\)

More recent and sophisticated methods have documented impaired expression of genes related to the formation of blood cells and Tumor Necrosis Factor (TNF\(\alpha\)) signaling in the peripheral blood of individuals with OUD.\(^\text{197}\) Tumor Necrosis Factor is an inflammatory cytokine that been considered as an anti-cancer agent. Reduced expression suggests reduced immune function in people with OUD. These researchers also found up-regulation of mitochondrial genes and splicing related genes, which are critical for generating different functional transcripts of the same gene.\(^\text{198}\) These are biomarkers of physical dependence on heroin, and the biomarkers overlap with impairment seen in people with other neurodegenerative disorders.\(^\text{199}\)

While none of what I outlined above is controversial, some still argue that addiction is not a biological or brain disease. The main reasons given are 1) that the first decisions to use drugs are largely voluntary, and 2) not everyone who uses drugs will ultimately become addicted. However, this fails to recognize the importance of biological and environmental risk factors in disease. Just as many people smoke who do not develop lung cancer, or many people eat too many carbohydrates do not develop diabetes, not everyone who uses drugs will develop physical dependence and addiction. The differing results can be explained in part by our unique genetic predisposition to SUD.

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\(^\text{194}\) Mei Zhu, et al., *Heroin Abuse Results in Shifted RNA Expression to Neurodegenerative Diseases and Attenuation of TNF gamma Signaling Pathway*, 8 *Scientific Reports* 1, 5 (2018)


\(^\text{198}\) Mei Zhu, et al., *Heroin Abuse Results in Shifted RNA Expression to Neurodegenerative Diseases and Attenuation of TNF gamma Signaling Pathway*, 8 *Scientific Reports* 1, 8 (2018) This analysis was conducted by comparing the RNA-seq data with healthy controls and people with OUD.

\(^\text{199}\) Mei Zhu, et al., *Heroin Abuse Results in Shifted RNA Expression to Neurodegenerative Diseases and Attenuation of TNF gamma Signaling Pathway*, 8 *Scientific Reports* 1, 8 (2018)
2. It is Not Just that Addiction Changes the Brain, Our Brains Can Predispose Us to Addiction

There is no magic dose or duration of opioid use that will predictably result in SUD. This is because not everyone is born with the same neuro-genetic risk factors, and not everyone experiences the same levels of childhood trauma or stress. Addiction has both genetic and environmental influences, similar to many other complex and chronic diseases.\(^{200}\) The genetic contribution to SUD is substantial, and accounts for somewhere between 40 and 50% of the risk associated with addiction.\(^{201}\) Clinical studies have shown that the heritability rates of opioid addiction are “similar to those of diabetes, asthma, and hypertension.”\(^{202}\) Additionally, because of the “enhanced neuroplasticity of their brains and their underdeveloped frontal cortex, which is necessary for self-control,” adolescents are at an increased risk of developing SUD.\(^{203}\)

There are many ways we can learn about the genetic contributions to traits. A conventional method looks to family and twin epidemiological studies, where we expect to see higher rates of addiction in homozygotic twins than in heterozygous twins, and more in both than we see in siblings. These studies show heritability estimates ranging from 30-60%.\(^{204}\) The variation is large because some genetic variants are common to all addictions and some affect risk to only a particular drug.\(^{205}\) For example, while twin studies show that SUD risk is shared among multiple classes of drugs, some genetic risk is specific to a class of drugs, such as opiates.\(^{206}\) Further, each stage of SUD will be impacted differently by genetics and the environment. The transitions from initiation of drug use to routine drug use, to physical dependence and even relapse may be driven by different genetic factors, as they involve different physiological processes.\(^{207}\)

\(^{200}\) Kenneth Blum et al., Genetic Addiction Risk Score (GARS), a predictor of vulnerability to opioid dependence, 10 FRONT. IN BIOSCIENCES 175, 177 (2018).


\(^{202}\) Nora D. Volkow and A. Thomas McLellan, Opioid Abuse in Chronic Pain — Misconceptions and Mitigation Strategies 374 NEW ENGL. J. MED. 1253, 1257 (2016)

\(^{203}\) Nora D. Volkow and A. Thomas McLellan, Opioid Abuse in Chronic Pain — Misconceptions and Mitigation Strategies 374 NEW ENGL. J. MED. 1253, 1257 (2016)

\(^{204}\) Mary Jeanne Kreek et al., Genetic influences on impulsivity, risk taking, stress responsivity and vulnerability to drug abuse and addiction, 8 NATURE NEUROSCIENCE 1450, 1450 (2005)

\(^{205}\) Mary Jeanne Kreek et al., Genetic influences on impulsivity, risk taking, stress responsivity and vulnerability to drug abuse and addiction, 8 NATURE NEUROSCIENCE 1450, 1450 (2005)

\(^{206}\) Mary Jeanne Kreek et al., Genetic influences on impulsivity, risk taking, stress responsivity and vulnerability to drug abuse and addiction, 8 NATURE NEUROSCIENCE 1450, 1450 (2005)

\(^{207}\) Mary Jeanne Kreek et al., Genetic influences on impulsivity, risk taking, stress responsivity and vulnerability to drug abuse and addiction, 8 NATURE NEUROSCIENCE 1450, 1450 (2005)
In addition to twin studies, researchers are using several novel techniques to discover the patchwork of genes involved in addiction generally, and OUD in particular. In the early 1970s, there was a “game-changing” discovery that opiate drugs bind to receptors in the brain, commandeering the endogenous internal system for reducing pain. There are three G protein-coupled receptors (GPCRs) in the opioid system, known as mu-, delta- and kappa-opioid receptors (MORs, DORs and KORs, respectively). Under normal conditions, these receptors are stimulated by endogenous opioid peptides, such as β-endorphin, enkephalins and dynorphins, that may be triggered in response to physical exertion or pain. However, when someone takes illicit drugs like heroin or pain medications like hydrocodone, these drugs easily bind to the same mu-opioid receptors, triggering a cascade of pain relief, pleasure, and dependence.

Recent research into the varying roles of the opioid receptors have yielded new insights into their crucial roles in regulating the opponent-processes of pain and pain relief. Opioid receptors play a role in each of the three steps of SUD. Activation of the MOR during intoxication triggers pleasure, or what was referred to as the α-process. Repeated MOR activation leads to reduced drug reward (tolerance) and dependence or withdrawal symptoms. KORs trigger the β-process of dysphoria through dynorphin, which characterizes withdrawal and abstinence. Stress and drug abuse both enhance KOR–dynorphin signaling. The DOR regulates reinforcement learning and memory, while also reducing anxiety and depressive states. In the relapse stage, evidence supports a role for DORs in context learning and memory for drug cues, like drug paraphernalia or drug-using friends. All three opioid receptors likely influence the preoccupation and craving state, and are implicated in drug-biased motivation, habit formation and loss of inhibitory control. Support for these roles comes from locations in the mouse brain with high receptor density, but these aspects are less well-characterized in the human brain.

208 Emmanuel Darcq and Brigitte Lina Kieffer, Opioid Receptors as “Drivers” of the Onset, Progression, and Maintenance of Addiction, 19 NATURE REVIEWS NEUROSCI 499, 504 (2018)
209 Emmanuel Darcq and Brigitte Lina Kieffer, Opioid Receptors as “Drivers” of the Onset, Progression, and Maintenance of Addiction, 19 NATURE REVIEWS NEUROSCI 499, 504 (2018)
210 Emmanuel Darcq and Brigitte Lina Kieffer, Opioid Receptors as “Drivers” of the Onset, Progression, and Maintenance of Addiction, 19 NATURE REVIEWS NEUROSCI 499, 504 (2018)
211 Emmanuel Darcq and Brigitte Lina Kieffer, Opioid Receptors as “Drivers” of the Onset, Progression, and Maintenance of Addiction, 19 NATURE REVIEWS NEUROSCI 499, 504 (2018)
212 Emmanuel Darcq and Brigitte Lina Kieffer, Opioid Receptors as “Drivers” of the Onset, Progression, and Maintenance of Addiction, 19 NATURE REVIEWS NEUROSCI 499, 504 (2018)
213 Brain areas associated with planning such as the prefrontal cortex, have moderate density of all types of receptors. MORs in the VTA trigger release of dopamine, projecting to the nucleus accumbens, which has a high density of MOR receptors and KOR receptors. The nucleus accumbens has a large role in reward-learning and registering the salience of drugs during intoxication. The
Given these unique receptor properties, targeting specific opioid receptors, by blocking KOR and activating DOR, is a promising target for the treatment of addiction, and other disorders like depression that stem from low reward or high-aversion states. Interestingly, when researchers deleted the OPRM1 gene in mice, this “simultaneously eliminated the analgesic, rewarding and dependence-inducing effects of morphine, demonstrating that the MOR is the sole responsible receptor for both the therapeutic and the adverse actions of morphine.”

Mutations in this receptor gene have been repeatedly associated with increased addiction risk. Recent research suggests there is substantial overlap between the genetic correlates of opioid addiction and pain sensitivity, with one team positing that mutations in the mu-opioid receptors might actually be the drivers of not just OUD, but other forms of SUD.

Of course, there are different pathways to addiction. There is no “one gene” for addiction risk. Despite the significant role of mutations in the opioid receptors, mutations in other genes also contribute to addiction risk. Genome-wide association studies (GWAS) have found significant associations between mutations linked with potassium and calcium signaling networks in the brain, and developing OUD. These results were most profound in the African-American subgroup sample. Glutamate, an excitatory neurotransmitter, also plays a large role in addiction. The N-methyl-d-aspartate (NMDA) glutamate receptor gene GLUN3A has been shown to serve an important role in the development of addiction.

Amygdala likewise has a high density of MOR, and to a lesser extent, KOR receptors. The MOR receptors in the amygdala promote pleasure, while the KOR receptors in the amygdala promote anxiety. The hippocampus has a high density of MOR and KOR receptors, and is critical in memory formation. See, Emmanuel Darcq and Brigitte Lina Kieffer, *Opioid Receptors as “Drivers” of the Onset, Progression, and Maintenance of Addiction*, 19 Nature Reviews Neuroscience 499, 504 (2018)

Specifically, mutations on the OPRM1; OPRK1; OPRD1; PDYN; POMC; PENK receptor genes have all been associated with increased risk of OUD. See, Kenneth Blum, et al., *Genetic Addiction Risk Score (GARS), a predictor of vulnerability to opioid dependence*, 10 Front. In Biosciences 175, 179 (2018).

*The[se] genes involved in the addictive process can also be indicative of which genes are engaged in pain mechanisms, pain sensitivity, and opiate addiction.” See, Kenneth Blum, et al., *Genetic Addiction Risk Score (GARS), a predictor of vulnerability to opioid dependence*, 10 Front. In Biosciences 175, 179 (2018)


Mutations on the GABA receptors have also been implicated, through microarray, single-gene strategies, and genome-wide association studies.\textsuperscript{221}

Due to the heavy involvement of dopaminergic pathways, mutations on the dopamine receptor gene, D2, (located on chromosome 11 q22-q23) have been extensively studied.\textsuperscript{222} Hundreds of studies have connected the DRD2 gene in particular to OUD, suggesting low baseline levels of hedonias and higher levels of anxiety as the behavioral phenotypes that give rise to abuse.\textsuperscript{225} One lab has determined that the DRD2 A1 mutation has a Positive Predictive Value (PPV) of 74%, indicating “that if a child is born with this polymorphism they have a very high risk of becoming addicted to either drugs, food, or aberrant behaviors at some point in their future.”\textsuperscript{224}

In addition to the substantial and growing evidence of genetic contributions to SUD, there is extensive research on the genetic risk for behavioral endophenotypes of addiction. For example, traits such as impulsivity, risk-taking and depression may contribute to the initiation of drug use as well as the transitions from initial use to regular use to addiction. As Robbins’ team has documented, individuals with hypodopaminergic systems (reduced endogenous dopamine release) and impaired inhibitory control in the cerebral cortex are vulnerable to developing SUD.\textsuperscript{225} Impulsive rats are not only much more likely to escalate self-administration of cocaine but also much more likely to relapse to a drug-seeking habit after some period of abstinence.\textsuperscript{226} People with depression\textsuperscript{227} and anxiety are also at increased risk of SUD. Each of these personality dimensions, or endophenotypes, has its own complex genetic basis.\textsuperscript{228}

Given that a significant risk factor for developing addiction lies in our genes, one lab has gone so far as to create a Genetic addiction risk score

\textsuperscript{221} Kenneth Blum, et al., Genetic Addiction Risk Score (GARS), a predictor of vulnerability to opioid dependence, 10 FRONT. IN BIOSCIENCES 175, 178 (2018).
\textsuperscript{222} Kenneth Blum, et al., Genetic Addiction Risk Score (GARS), a predictor of vulnerability to opioid dependence, 10 FRONT. IN BIOSCIENCES 175, 177 (2018).
\textsuperscript{223} Kenneth Blum, et al., Genetic Addiction Risk Score (GARS), a predictor of vulnerability to opioid dependence, 10 FRONT. IN BIOSCIENCES 175, 178 (2018).
\textsuperscript{224} Kenneth Blum, et al., Genetic Addiction Risk Score (GARS), a predictor of vulnerability to opioid dependence, 10 FRONT. IN BIOSCIENCES 175, 181, 186 (2018)
\textsuperscript{225} Christopher Evans and Catherine Cahill, Neurobiology of Opioid Dependence in Creating Addiction Vulnerability, 5 F1000 RESEARCH 1748, *3 (2016)
\textsuperscript{226} BARRY EVERITT, ET AL., NEURAL MECHANISMS UNDERLYING THE VULNERABILITY TO DEVELOP COMPULSIVE DRUG-SEEKING HABITS AND ADDICTION, 363 Philosophical Trans. of the Royal Society 3125, 3130 (2008)
\textsuperscript{227} Mark Edlund, et al., Opioid Abuse and Depression in Adolescents: results from the National Survey on Drug Use and Health, 152 DRUG AND ALCOHOL DEPENDENCE 131, 131 (2018)
\textsuperscript{228} Mary Jeanne Kreek et al., Genetic influences on impulsivity, risk taking, stress responsivity and vulnerability to drug abuse and addiction, 8 NATURE NEUROSCIENCE 1450, 1450 (2005)
It is hoped that these risk scores, calculated based on the presence of multiple mutations, can better predict the development of SUD, and relapse. These tools may one day be used in the clinic, as a screening tool for prescribing addictive medications like opioids, or to help prevent problem drug-use before it rises to the level of dependence.

3. Genetic Explanations Might Reduce Responsibility, But Do Not Reduce Stigma on Their Own

We now know that addiction is caused in large part by our genes, which we have no control over and we cannot change. Given this, some have suggested that, “questions about right or wrong-doing seem to be ill-posed.”

This is because we typically blame people for things that are under their control, and for which they make a voluntary decision to do. It is much harder to justify blaming someone for their behavior, once we know that for genetic reasons, it is considerably more difficult for them to conform their behavior to a particular standard. This presents some challenges to the pure moral choice model of addiction, as there are clear inequities in our inherent abilities to refrain from becoming dependent.

Do we blame people for developing PTSD or Alzheimer’s? And even if we continue to blame someone for developing lung cancer or diabetes, because these too are caused in part by behavioral choices, we still view these diseases as a medical problem deserving of a medical treatment. It would be far too ambitious to expect the IDM to erase stigma completely. Rather, the IDM model will be more effective at helping policymakers and the public understand that some people are much more vulnerable to addiction, due to their neuro-genetics. This would hopefully lead to policies that emphasize treatment, prevention, and harm-reduction, rather than criminalization and social isolation. Addiction is a disease, we should treat it like one. While we can and must address social and personal determinants, we must initially focus primarily on the primary determinants of health, as we would with any other disease.

Proponents of the brain disease model have argued that a neuroscience perspective reduces attributions of free will, (namely volition

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229 See, Kenneth Blum, et al., Genetic Addiction Risk Score (GARS), a predictor of vulnerability to opioid dependence, 10 FRONT. IN BIOSCIENCES 175, 179 (2018).
230 Lily Frank and Saskia Nagel, Addiction and Moralization: the role of the underlying model of addiction, 10 NEUROETICS 129, 132 (2017)
231 “[B]oth those in favor of and those opposing the brain disease model of addiction appear to be in agreement about the actual existence of an effect of neuroscience information on belief in free will; otherwise, the debate would be moot.” Eric Racine, Sebastian Sattler, and Alice Escande, Free Will and the Brain Disease Model to Addiction: The Not So Seductive Allure of Neuroscience and Its Modest Impact on the Attribution of Free Will to People With an Addiction, 8 FRONTIERS IN PSYCHOL. 1850, 1853 (2017)
and responsibility) because it “relocates the disorder to the brain, rather than to the person.” However, as mentioned infra at [x], the disease need not be “located” in the brain to be an ordinary disease worthy of treatment. In keeping with this general idea, the National Alliance on Mental Illness (NAMI) argued that once the public understood mental illnesses to be a “real,” disease, and with a similar biological etiology to cancer or diabetes, prejudice and discrimination would fade. Their campaign, “A Disease Like Any Other,” focused on educating people about the brain disease model of mental illnesses such as addiction. National surveys have documented the success of their educational messaging, with mental health literacy increasing significantly in recent decades. Many more Americans now appreciate that substance abuse has a large hereditary and environmental component, and is not entirely a disease of the weak-willed.

However, importantly, so far this has not lead to universal, reduced attributions of stigma. Indeed, attributing addiction to genetic factors, may have caused some backlash. This may be because genetic causes appear immutable and with lasting impact for generations.

Two meta-analyses, looking at the effect of the brain disease model on attributions of responsibility and stigma yielded consistent patterns. When the team reviewed the experimental studies, they indicated that neuro-genetic explanations “reduced blame, increased perceived dangerousness and prognostic pessimism, and had no effect on social distance.” The review of the correlational studies found that “people who endorse biogenetic explanations tend to blame affected persons less for their problems, but perceive them as more dangerous and desire greater

232 Eric Racine, Sebastian Sattler, and Alice Escande, Free Will and the Brain Disease Model fo Addiction: The Not So Seductive Allure of Neuroscience and Its Modest Impact on the Attribution of Free Will to People With an Addiction, 8 FRONTIERS IN PSYCHOL. 1850, 1850-51 (2017)
233 Bernice Pescosolido, The Public Stigma of Mental Illness: what do we think; what do we know; what can we prove? 54 J. Health and Social Behavior 1, 11 (2013)
234 Bernice Pescosolido, The Public Stigma of Mental Illness: what do we think; what do we know; what can we prove? 54 J. Health and Social Behavior 1, 11 (2013)
235 Amy Loughman & Nick Haslam, Neuroscientific explanations and the stigma of mental disorder, a meta-analytic study, 3 Cognitive Research: Principles and Implications, 1, 2 (2018)
236 Bernice Pescosolido, The Public Stigma of Mental Illness: what do we think; what do we know; what can we prove? 54 J. Health and Social Behavior 1, 11 (2013)
237 Phelan has suggested this “backlash,” as “genetic attribution …significantly associated with higher levels of stigma, …because the mark conveyed a sense of permanence, having effects that live on in families past the time of any one individual.” See, Bernice Pescosolido, The Public Stigma of Mental Illness: what do we think; what do we know; what can we prove? 54 J. of Health and Social Behavior 1, 11 (2013)
239 Amy Loughman & Nick Haslam, Neuroscientific explanations and the stigma of mental disorder, a meta-analytic study, 3 Cognitive Research: Principles and Implications, 1, 2 (2018)
social distance from them." No correlational studies addressed prognostic pessimism. These findings led the researchers to propose the “mixed blessings” model of stigma. Fueled in part by a belief that a “deep seated, hidden essence is shared by all members of a category” the brain disease model reduced individual control and therefore moral responsibility, but increased genetic essentialist thinking, or that affected people have predictable traits that can be explained almost entirely by their genes. Essentialist thinking leads to fear, social distance, and pessimism about treatment, as the genes are given a powerful role in determining behavior. When we think of people as having a difference, that is immutable, can be labeled, and that leads to antisocial behavior, this can engender the very stigma and dehumanization we sought to avoid.

In contrast, people may think of neurobiological causes of disorders as less binary than genetic causes, as brains are less static, and more plastic. Genes may be thought of as deep, ultimate causes, with the brain operating at a more intermediate level of translation. A study focusing more on the neuroscientific explanations of mental illness generally, found similar results to the genetic studies. However, as an author of one of the studies included in the analysis, the authors could not conclude that our study isolated neuroscientific explanations, as our causal model included genetics and neuroscience explanations for the aberrant behavior. Nonetheless, the authors concluded that it could be that people misunderstand how the brain works and think of it deterministically like genes. Or, it could be that understanding someone as having reduced behavioral control makes them more unpredictable, and therefore, scarier and deserving of punishment.

A recent experiment sought to explore the impact of neuro-genetic causal models of addiction on attributions of free will and responsibility. The researchers showed respondents text and a neuro-image (which unfortunately explained the neurobiological basis of addiction by

240 Amy Loughman & Nick Haslam, Neuroscientific explanations and the stigma of mental disorder, a meta-analytic study, 3 COGNITIVE RESEARCH: PRINCIPLES AND IMPLICATIONS, 1, 2 (2018)
241 Amy Loughman & Nick Haslam, Neuroscientific explanations and the stigma of mental disorder, a meta-analytic study, 3 COGNITIVE RESEARCH: PRINCIPLES AND IMPLICATIONS, 1, 3 (2018)
242 Amy Loughman & Nick Haslam, Neuroscientific explanations and the stigma of mental disorder, a meta-analytic study, 3 COGNITIVE RESEARCH: PRINCIPLES AND IMPLICATIONS, 1, 3 (2018)
243 I Dar-Nimrod and S. J. Heine, Genetic essentialism: on the deceptive determinism of DNA, 137 PSYCH. BULLETIN 800, 2011) “Genes are popularly understood to be discrete, hidden, fixed, and identity-determining to the point where DNA has become a colloquial synonym for essence.” Dar-Nimrod and Heine have reviewed the socially destructive implications of genetic essentialism (for example, with racism).
244 Amy Loughman & Nick Haslam, Neuroscientific explanations and the stigma of mental disorder, a meta-analytic study, 3 COGNITIVE RESEARCH: PRINCIPLES AND IMPLICATIONS, 1, 3 (2018)
245 Amy Loughman & Nick Haslam, Neuroscientific explanations and the stigma of mental disorder, a meta-analytic study, 3 COGNITIVE RESEARCH: PRINCIPLES AND IMPLICATIONS, 1, 7 (2018)
246 Amy Loughman & Nick Haslam, Neuroscientific explanations and the stigma of mental disorder, a meta-analytic study, 3 COGNITIVE RESEARCH: PRINCIPLES AND IMPLICATIONS, 1, 7 (2018)
referencing reduced dopamine receptors in people who have SUD, which could be an effect of the disorder rather than a cause), and compared this with a control group. The respondents were asked to indicate the extent to which they agreed with statements about cocaine addicts’ and alcoholics’ diminished free will. The findings were modest, but found attributions of volition were somewhat reduced for the cocaine subsample, when respondents viewed the textual and neuro-image explanations together, but there was no effect when respondents viewed the text or neuro-image alone, or when the information applied to alcoholics. However, respondent characteristics such as education and self-reported knowledge of neuroscience were associated with lower attributions of responsibility for both substances, and education was associated with lower attribution of volition for the alcohol sub-sample.

In summary, the brain disease model has not been an unmitigated success. There are risks associated with explaining addiction in terms of neuroscience, as “descriptive neuroscience concepts are inseparable from historical attitudes and intuitions towards addiction and addicted persons. Placing emphasis on the diseased brain may foster unintended harm by paradoxically increasing social distance towards the vulnerable group the term is intended to benefit.” However, it has moved the needle on pushing the public to support more treatment. While endorsements of a genetic cause of addiction has led to greater support for seeking treatment from psychiatrists, hospitals, and medications, in some cases it has also provoked greater cynicism about the potential efficacy of treatment. Clearly, the brain disease model of addiction, relying solely on neurogenetics, cannot work alone.

F. We Must Develop a New Public Health Campaign Based on the IDM

The brain disease model of addiction, with a heavy emphasis on the idea that the brain is “hijacked” and the genes are determined, has not

247 Eric Racine, Sebastian Sattler, and Alice Escande, Free Will and the Brain Disease Model of Addiction: The Not So Seductive Allure of Neuroscience and Its Modest Impact on the Attribution of Free Will to People With an Addiction, 8 FRONTIERS IN PSYCHOL. 1850, 1850-51 (2017)
248 Eric Racine, Sebastian Sattler, and Alice Escande, Free Will and the Brain Disease Model of Addiction: The Not So Seductive Allure of Neuroscience and Its Modest Impact on the Attribution of Free Will to People With an Addiction, 8 FRONTIERS IN PSYCHOL. 1850, 1850-51 (2017)
249 Eric Racine, Sebastian Sattler, and Alice Escande, Free Will and the Brain Disease Model of Addiction: The Not So Seductive Allure of Neuroscience and Its Modest Impact on the Attribution of Free Will to People With an Addiction, 8 FRONTIERS IN PSYCHOL. 1850, 1850-51 (2017)
251 Bernice Pescosolido, The Public Stigma of Mental Illness: What Do We Think; What Do We Know; What Can We Prove? 54 J. HEALTH AND SOCIAL BEHAVIOR 1, 11 (2013).
worked so far to reduce stigma and increase treatment. What is needed is a more nuanced model, such as the IDM, that can confront the reality of addiction as a disease, with neuro-genetic and environmental risk factors which exist on a continuum, and for which there are successful treatments. Recent data from public health suggests that when combined in this way, the neuro-genetic evidence can work to reduce stigma. Rather than seeing the brain as hijacked and controlling the disease process entirely, the IDM can explain important causes of addiction as neuro-genetic, while recognizing the importance of environmental risk and personal choice. We need to stop feeding the false dichotomy of disease or choice, with a more nuanced public health campaign that is experimentally tested and uses destigmatizing language and images.252

The false dichotomy has led some physicians to flip back and forth between the moral choice and disease model, when explaining addiction to their patients.253 Studies demonstrate that physicians feel the need to switch back and forth between “their deployment of disease, moral and social models depending on how they wish to frame a client’s sense of responsibility for the problem and the solution...” 254 Rather than asking physicians to be agile, and selectively employ different models of addiction, the IDM allows them to speak with one consistent message.255 There are not competing models of addiction—there is one model that accommodates developments in neuro-genetics and psychology, and reflects the reality of people living with addiction, that it involves some level of individual choice, and leads to antisocial behaviors that we would like to see treated. The only reason this simple resolution has been so hard to grasp in the field of addiction, and not elsewhere, is the pervasiveness of the stigma surrounding the disease.

a. Part 1 of the Public Health Campaign: Addiction Risk Exists on A Continuum, And Presents as a Neurobiological Disease

So far, the “dialogue around opioids has been dominated by several

253 Lily Frank and Saskia Nagel, Addiction and Moralization: the role of the underlying model of addiction, 10 Neuroethics 129, 133 (2017)
approaches that on their own are inadequate or harmful.” To mitigate stigma, we must develop a comprehensive public health campaign that is based on a model like the IDM. This campaign should rely on individual stories as well as data, to explain the simple facts that anyone—no matter their education, class, or race—can develop SUD. Using the neurobiological evidence for SUD, the public health message must also include information that once someone is addicted, their voluntary choices are highly constrained because of the disease. The specific messaging of the campaign should be created after experimental testing. However, recent studies from public health make it clear that certain types of messages will be more successful than others to reduce stigma and encourage treatment.

The first way we can reduce the “othering” of people with addiction is to emphasize that the disease, and its risk factors, exist on a continuum. While some were concerned that a model of addiction that focused on the brain would encourage an “Us vs. Them” dynamic, (the normal and the diseased), we can diminish this effect by instead by focusing on the idea that risk factors are not categorical, and each of us has varying degrees of genetic risk.

People with SUD are not categorically different from us. Rather, they have underlying genetic and environmental vulnerabilities, and these risks are present to a degree in each of us. With the right combination of factors, any one of us can be affected. Addiction is not a disease of the weak or immoral. Public health campaigns should emphasize that there is an underlying vulnerability in each of us that we did not cause and cannot control. As there are different pathways to addiction, some of us are at greater risk due to our genes for processing opioids, genes for processing dopamine, or genes for memory encoding, etc. If you prefer to speak in terms of endophenotypes, some of us are at greater risk due to our anxiety, depression, or impulsivity. This is consistent with the Research Domain Criteria put forward by the National Institutes of Health, which advocates

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256 Brendan Saloner, et al., A Public Health Strategy for the Opioid Crisis, 133 Public Health Reports 24S, 26S (2018)(“War on drugs’ approaches that would increase arrests and incarceration to deter drug use and distribution have had long-term scarring effects on many communities, primarily those of color, without measurably reducing access to street drugs. Likewise, defining drug use as an individual’s moral failing that can be remedied through willpower alone is inconsistent with biological triggers that create susceptibility to addiction. The moral failing approach also fails to recognize the role of trauma and adverse childhood experiences in addiction.”)


260 As all psychological constructs are all mediated by the brain, the IDM does not replace these models, but incorporates them.
for understanding mental illness as a constellation of component psychological and neurobiological processes, which exist on a continuum. Each mental illness can be conceived of as a natural process, such as reward learning or fear processing, that has become extremely disordered. But we all have varying levels of disorder in different domains.

This non-categorical way of thinking is in keeping with the new Diagnostic and Statistical Manual (DSM-V) diagnostic criteria for SUD, which recognizes that SUD exists on a continuum. This is not only the right way of thinking about SUD, in terms of our underlying neuro-genetic risk factors. It will also reduce the extent to which we think of people with SUD as the categorical “other,” which can lead to stigma and dehumanization.

Viewing SUD as existing on a continuum can also help people get treatment sooner. It is difficult to remove the stigma from addiction because people conflate severe SUD with all forms of SUD. People do not get treatment because they do not want to accept the label of “addict,” because in their minds there is only recreational drug use, and extreme, full-blown “addiction.” There is nothing in the middle. Getting people to access treatment when the disease is in its modest or early stages will lead to much better recovery outcomes. Just as lung cancer patients have much better prognoses if the cancer is caught at Stage 2 rather than at Stage 4, so too do patients with modest SUD fair better than people with severe SUD.

b. Part 2 of the Public Health Campaign: Addiction is Treatable

When reporting on addiction, journalists often focus on specific individuals, even when the highlighted individual is atypical. The public then extrapolates from this narrative, which impacts how they view the entire affected population. Most U.S. news media coverage of opioid analgesic abuse from 1998 to 2012 focused on illegal drug dealing and over-prescribing of pain medications by physicians. Among the news stories that mentioned a solution, law enforcement arrests and punishment...
were mentioned most frequently, at 64% of news stories, with only 3%
mentioning expanding substance use treatment and less than 1% suggesting
harm-reduction policies.\textsuperscript{266} In keeping with the moral choice model of
addiction, news stories emphasized OUD as a criminal issue, rather than as
a treatable medical condition.\textsuperscript{267} This has exacerbated stigma, as people
think of SUD as an untreatable condition. This may also be why 65% of
Americans thought people with untreated alcoholism were likely to be
violent, and 87% thought someone with untreated cocaine dependence were
likely to be violent, even though they are much more likely to injure
themselves.\textsuperscript{268} Stories that depict addicts as dangerous are more likely to
lead to stigmatized views and a sense that these people need to be punished.

Newsflash: \textit{most} people who are treated for SUD with evidence-
based treatments achieve remission.\textsuperscript{269} This fact needs to be much more
widely known. We need to fund public health campaigns that emphasize
treatment options, and the efficacy of those options.\textsuperscript{270} A few recent studies
demonstrate that when news media mentions treatability, this is positively
correlated with endorsing mental health treatment policies.\textsuperscript{271} Further, when
drug addiction is portrayed as a treatable health condition, this reduces
desire for social distance, improves belief in the effectiveness of treatment,
and lessens willingness to discriminate against people with SUD.\textsuperscript{272}
Specifically, as compared to respondents who read vignettes about
untreated OUD, when respondents read vignettes about individuals with
SUD who had been successfully treated, this group was much less likely to
reject the prospect of working with someone with addiction or having them
marry in to the family.\textsuperscript{273} Reading about individuals in recovery also made

\textsuperscript{266} Emma McGinty, et al., \textit{Criminal Activity or Treatable Health Condition? News Media Framing of
Opioid Analgesic Abuse in the United States}. 67 Psychiatric Services 405, 408 (2016)
\textsuperscript{267} Emma McGinty, et al., \textit{Criminal Activity or Treatable Health Condition? News Media Framing of
Opioid Analgesic Abuse in the United States}. 67 Psychiatric Services 405, 408 (2016)
\textsuperscript{268} Emma McGinty, et al., Portraying mental illness and drug addiction as treatable health
conditions: effects of a randomized experiment on stigma and discrimination. 126 Soc Sci Med. 73,
74 (2015); Bernice Pescosolido, et al., \textit{"A Disease Like Any Other." A decade of change in public
reactions to schizophrenia, depression, and alcohol dependence}, 167 Am. J. Psych. 1321, 1321
(2010); see also CL Barry, et al, After Newtown—public opinion on gun policy and mental illness,
\textsuperscript{269} Sarah Wakeman, et al., \textit{OPIOID USE DISORDER, STIGMA, AND TRANSPLANTATION: A CALL TO
\textsuperscript{270} McGinty, et al., 69 Psychiatric. Serv. 136 (2017)
\textsuperscript{271} Emma McGinty, \textit{ET AL.}, \textit{TRENDS IN NEWS MEDIA COVERAGE OF MENTAL ILLNESS IN THE UNITED
\textsuperscript{272} Emma McGinty, et al., \textit{Portraying mental illness and drug addiction as treatable health
conditions: effects of a randomized experiment on stigma and discrimination. 126 Soc Sci Med. 73,
73 (2015); Daniel Romer, Reducing the stigma of mental illness among adolescents and young
\textsuperscript{273} Emma McGinty, et al., \textit{Portraying mental illness and drug addiction as treatable health
conditions: effects of a randomized experiment on stigma and discrimination. 126 Soc Sci Med. 73,
78-79 (2015);
respondents more likely to believe treatment can effectively control symptoms, though this particular study did not find support for increased spending on addiction treatment, as older studies have.274

IV. Conclusion

The IDM I propose recognizes that SUD is not a moral failing, but a complex and chronic disease, with environmental and neuro-genetic risk factors. Rather than furthering the false dichotomy between moral choice and biological disease, the IDM places addiction on equal footing with other complex and chronic diseases, such as lung cancer or diabetes, each of which has environmental and genetic risk factors. Through the IDM, the policy and public health emphasis can be properly placed on treatment. Given that addiction is a medical problem, our primary response should be medical, not criminal—and not even sociological. While we should not ignore important social determinants of health, such as childhood trauma, access to safe housing and jobs, and criminal justice reform, these should be secondary concerns. Addiction is a disease, but due to rampant dehumanization of people with addiction, we have failed to see it as such. The stigma surrounding people with SUD is rampant, and will take concerted effort to mitigate. By engaging in a massive public health campaign that emphasizes that addiction risk is neuro-genetic and exists on a continuum, that once addiction takes hold voluntary choices related to drug use are constrained, and that treatment can be quite effective, we can start telling a different, and more hopeful story about recovery. As stigma in its many forms is a major obstacle in the treatment of addiction, mitigating stigma will have a cascade of positive effects. Reducing stigma will encourage people to seek treatment, will help ensure that the treatment that they receive is evidence-based and compassionate, and will reduce the unfair discrimination and criminalization that people with SUD experience.

274 Emma McGinty, et al., Portraying mental illness and drug addiction as treatable health conditions: effects of a randomized experiment on stigma and discrimination. 126 Soc Sci Med. 73, 78-79 (2015);